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OLFACTORY IMAGINATION AND OLFACTORY HALLUCINATIONS

AN EXPERIMENTAL AND CLINICAL STUDY OF THE SENSE OF SMELL IN NORMAL AND IN PSYCHOTIC PERSONS

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This study is concerned with the subjective part of olfactory sensation. Recent psychiatric research [1] has stressed the importance of the study of the subjective aspects of perception in attempting to solve problems of psychiatry and theoretical psychology of the senses. The question of the mechanics of orientation in the external world, as well as the mechanics of illusions, hallucinations, etc., in patients with mental disease is involved in this problem. Contact with the world of reality in the last analysis depends on perception of sensation mediated by the five senses. Next to direct sensational images, the reality of which is tacitly assumed in consciousness, after-effects of sensations possess the character of reality immediately associated with perception. Eidetic images (Jaensch [2]) and memory images (representations), although their realness as compared with actual images is less, can and often do have a character of reality close to that of the sensory image. Thus the varying categories of contact which the perceptive self has with the external world are illustrated in these types of perceptive experiences. Further, the meaning of these phenomena to the personality varies in relation to the consciousness, emotional constellations and psychic, structural and physical dispositions of the individual.

The present study follows others which have dealt with perception of other senses. Kanner and one of us (P. S.)[3] examined optic

From the Research Department of the Psychiatric Division of Bellevue Hospital.

1. Schilder, Paul: Experiments on Imaginations, After-Images and Hallucinations, Am. J. Psychiat. **13**:597 (Nov.) 1933.

2. Wertham, Frederic: Progress in Psychiatry: Eidetic Phenomena, Arch. Neurol. & Psychiat. **24**:809 (Oct.) 1930.

3. Kanner, L., and Schilder, P.: Movements in Optic Images and the Imagination of Optic Movements, J. Nerv. & Ment. Dis. **72**:489, 1930.

imagination (representations) and optic images of normal persons. It was found that optic images occurring after representation had properties and behaved like after-effects on one hand, and like eidetic images on the other. Tendencies to movement (rhythmic waving, scintillation, etc.) were striking in the optic images. In the tactile sphere, we [4] found changes of a similar nature in the represented images (imagination) of tactile sensation; this was valid also for tactile after-effects. Movements, diffusion, fragmentation and other types of perceptive disintegration of tactile experiences, noted in these experiments in imagination and after-effects, were similar to those seen in various lesions of nerves, the spinal cord and cortex, as tabes or lesions of the parietal lobe or mescal intoxication, in which tactual sensory disturbances exist. Among patients with alcoholic hallucinations, one of us (W. B.)[5] has shown that similar but intensified changes in tactile after-effects occur and that probably these speeded up perceptive tactile changes are related to the hallucinations of the alcoholic psychoses. On the other hand, in schizophrenia the changes in perception are negligible. In other words, the toxic influence in the cortex is represented by a "changed" perception in the tactile sphere. These results have lent support to the contention of one of us (P. S.) that disorders of sensation, which are in the foreground in pathologic conditions of the central nervous system, are in the background of experience in normal persons. In the field of auditory sensation a similar study is being completed at present by Parker and one of us (P. S.) in this clinic. The perception of gustatory sensation and imagination are dealt with in this communication, but as a secondary concern.

The paucity of olfactory studies from a psychologic or sensational point of view is in marked contrast to the enormous literature concerned with optic and tactual sensation. There have been several psychoanalytic studies, however, for Brill [6] and Daly and White [7] have written on the meaning of olfactory symptoms in neuroses and psychoses. Recently, Henning [8] and von Skramlik [9] discussed the

4. Bromberg, W., and Schilder, P.: On Tactile Imagination and Tactile After-Effects, J. Nerv. & Ment. Dis. **76**:1 (July) 1932.

5. Bromberg, W.: Tactual Perception in Alcoholism, Arch. Neurol. & Psychiat. **28**:37 (July) 1932.

6. Brill, A. A.: The Sense of Smell in the Neuroses and Psychoses, Psychoanalyt. Quart. **1**:7 (April) 1932.

7. Daly, C. D., and White, R. S.: Psychic Reactions to Olfactory Stimuli, Brit. J. M. Psychol. **10**:70, 1930.

8. Henning, Hans: Psychologische Studien am Geruch, in Abderhalden, E.: Handbuch der biologischen Arbeitsmethoden, Vienna, Urban & Schwarzenberg, 1924, sect. 6, pt. A, no. 4.

9. von Skramlik, E.: Handbuch der Physiologie der niederen Sinne, Leipzig, Georg Thieme, 1926, vol. 1.

problem from the angle of general psychology and physiology, and Boernstein [10] from the standpoint of pathologic processes in the brain. Perky,[11] in 1910, working in Titchener's laboratory, found on testing olfactory imagination in research students that movements of the muscles of expression occurred with imagination in 20 per cent of cases, while in experiencing actual odors movements were more frequent. He did not extend his investigation further, nor did he discuss the subjective side of the problem.

The present report deals with the problem of representations of smell wilfully produced. The method employed was to study the perception of imaginations of smell in normal persons. In order to appreciate this properly we studied the after-effects of smell and the phenomenology of smell itself; then we investigated taste in like manner. Von Skramlik stated that he could reproduce imagined smells of great intensity with effort even without optic representations. Some of Henning's subjects could produce imaginations of smell without producing visual images. Ribot [12] reported that 12 per cent of the persons to whom he sent questionnaires were able to imagine all kinds of smells.

EXPERIMENTAL MATERIAL

We examined twelve normal persons (mostly physicians) systematically and many other subjects in a less systematic way. The subjects were requested to report any type of sensory experience occurring, particularly that in the nose, mouth or head, when they were asked to experience or to smell odors. The subject was asked:

1. What is experienced in the nose when the subject does not breathe?

2. What is experienced when the subject breathes naturally?

3. What are the smell sensations during and after having smelled coffee (ground), perfume essence (verdi vera), tincture of iodine, tincture of benzoin, butyric acid?

4. What is the sensation of smell after imagining: the smell of a rose, of oranges, of frying bacon, of burning rubber, of urine and feces; a smell without a body (the smell of the sea; the smell of the forest; the smell of a room with cooking)?

5. What are the experiences in taste when not eating?

6. What tastes occur after eating cheese?

7. What sensations occur after imagining the taste of a sweet substance, a bitter substance, a food?

1. *Sensation in Nose When not Breathing.*—Most subjects felt the bridge of the nose and had definite tickling, itching, etc., and sensations around the nostrils, especially on the inside surface. A feeling of tension in the head, throat and

10. Boernstein: Ueber den Geruchsinn, Deutsche Ztschr. f. Nervenh. **104:** 55, 1928.

11. Perky, C. W.: An Experimental Study of Imagination, Am. J. Psychol. **21:**422, 1910.

12. Ribot, quoted by Henning.[8]

around the nose was common. There was no sensation of smell, however. Subject G. R. said: "I have the feeling I have to move the nostrils all the time." When one does not breathe, there is no sensation on the mucous membrane. If one does not breathe the nose is filled with equally distributed tepid air. When the breath is held the sensation of warmth increases. The air in the nose is definitely felt as something outside the body.

2. *Sensation Experienced While Breathing.*—When breathing through the mouth a general feeling of freshness was experienced in the mouth and pharynx. In breathing naturally through the nose the type of sensation experienced was uniform in all the subjects. The tepid reservoir of air in the nose was disturbed by the stream of air which came into the nose. The stream was localized in the middle of the nose, but there was a definite tactile sensation on the mucous membrane on the intake, which increased in degree the more sharply the air was drawn in. It was as if the shaft of fresh air penetrated the envelop of warm air that was lying along the mucous membrane, which had no smell in it but gave an impression best described as that of stale air. As subject Sc. put it: "the (incoming) air is felt as a sharp cool cylinder around which is the stale warm air belonging to the nasal cavity." Subject B described the blanket of air as being "warm" and "static." With the expiration of the air, there is, according to Subject S., a "generalized feeling of warmth throughout the nose." The inrushing air during breathing had for the observer the character of an immediate perception; it was treated as an objective experience.

3. *Sensations During Smelling and After-Effects of Smell.*—All the subjects were offered smelling substances for a reasonable period of time, placed in open containers about 10 cm. from the nose. The substances offered were coffee, perfume, medical articles and butyric acid.

The phenomenology of smell under ordinary circumstances has been studied by von Skramlik; our experiments have confirmed his findings. Von Skramlik showed that the stream of air has to pass into the nose when the patient smells. It does not matter whether the stream of the air goes from the nose to the choanae or in the opposite direction. It is not sufficient that the smelling substance is in the nose, even though it is there in great quantities. But it is necessary for the perception of smell that the stream of air carrying the odor go through the nose. The smelling substance must be below the anterior part of the opening of the nose. All subjects agreed in the tests that they perceive a "smelling" odorous object and that they try to localize this smelling object. Subject F, when presented with butyric acid, said: "I had a sharp smell at first in the anterior chamber of the nose . . . (later) it seemed as if the smell were on the upper lip along the nares, being fed from the moustache into the nose." The object is perceived as surrounded by smell; i. e., the smell is coming off the object and the substance in question is "odorous." Thus, subject Sc. said of butyric acid: "The smell appears to be a dense gas outside the nose"; again, Sc. said of coffee: "The smell is around the object and drawn into the nose—smell is in the air of the nose." Some subjects perceived the smell around the outside of the nose; subject F. said of coffee: "I have a feeling of vapor around the nose." The smell, then, goes from the object to the nose and is perceived as being in the nose. When the odor has left the smelling object, one is concerned then only with the odor and no longer with the substance. There is nothing in olfactory perception which involves the smelling object—it is not represented per se in the olfactory image. But the smell itself is perceived as

an object and it becomes the object for olfactory consciousness; one speaks then of an "object-smell."

In the smells of medium intensity which were used, after the first moment of experience the smell disappeared, but came back immediately. When forced breathing was attempted, the experience of smell was replaced by sensations of a "fresh" quality in the mucous membranes, because after the stream of air conveying the smell has passed through the stale tepid air in the nose, the latter appears to hold the smell with even a greater intensity than when it was carried by and in the incoming stream. Most of the subjects said that the smell goes only to the middle of the nose; it seems to fill out into the lower part of the nose. Only butyrin, which has more irritating qualities, was occasionally felt in the membranes. But this pungent sensation was distinctly different from smell and was allied to the so-called chemical sense-irritation. In this connection only one subject felt the smell as a "rain of particles." Sc. said: "I feel the odor distributed like sun-burst particles." In the situations in which the odor was combined with chemical irritation, that is, when the odor was experienced as being unusually pungent, the sensation of the mucous membranes was distinct: Subject D. said of tincture of iodine: "I felt a prickling sensation on the mucous membrane," and subject F. said of perfume: "It went straight up the nostrils. The smell is a trifle pungent and seems to penetrate the mucous membrane."

The fate of the object-smell as it streams toward the entrance of the nose depends on the current of air sniffed in by the subject. The object-smell may be situated in a cloud around the nostrils instead of coming off the object. The smell usually intrudes into the nostrils up to the middle of the nose. It may project farther into the nose when it is sufficiently strong or if drawn in deeply. Or it may be carried to adjacent regions as perceived sensation of other types. Thus, subject D. said of iodine: "The smell is carried to my palate where I have a taste reaction" and subject R. said: "The smell goes into my brow over the eyes." Subject R. said of tincture benzoin: "I have the feeling that the smell goes into my chest." Subject S. said of iodine: ". . . later the smell diffuses and goes into the nasopharyngeal area, but not so deeply or precisely as in the nose." By forced breathing the object-smell may disappear completely or may remain around the central shaft of air. Gentle breathing helps the appreciation of smell—it helps the object-smell to get into the nose. Plessner[13] was wrong when he expressed the belief that smell is localized on a mucous membrane, for as subject F. said of perfume: "The smell seemed to be a 'pencil' of smell which penetrated into the nasal cavity but was smaller than the cavity, i. e., did not fill it."

On the whole, it can be said that in the perception of smell there is, inherently, a knowledge of the smelling object. This knowledge may be represented by images or by the senses, which may be a complex synesthesia. The smell sensation fuses into smell memory. Subject A. said of butyric acid: "The smell became soft and pleasant as it thinned out. I smelt and thought of the distinct odor of St. John's bread which I have not seen for years. I could not separate the two smells."

Most psychologists, for example, Froebes,[14] have agreed that the smell apparatus fatigues rather quickly. In our experiments, using stimuli of comparatively small intensity, we found that smell persisted only a short time. In the duration

13. Plessner: Die Einheit der Sinne, Bonn, 1923, quoted by Henning.[8]

14. Froebes: Lehrbuch der experimentellen Psychologie, Heider-Freiburg, Heider, 1923, vol. 1, p. 132.

of smell, as well as in the mechanism of smell sensation, the factor of motor activity is important. Most investigators seem not to have placed sufficient emphasis on the importance of the motor components of smell. Boernstein for one denied (erroneously, we think) that smell has any motility. In our experiments this motor activity finds outspoken expression in the sensations. On smelling coffee subject M said: "I have a feeling of movement, originating in the bottle and going to the nostrils, with visual images of streams of coffee going into the nose." As von Skramlik showed, a stream of air is necessary to convey impressions of smell. It is a peculiarity of the sense of smell that it requires motor activity for its presentation for perception. This is seen in the reflex (automatic) respiratory movements of smelling, but even here some regulation of breathing is necessary for smelling activities; i. e., one has to sniff in order to get a sufficient quantity of the object-smell for perception. The object-smell, then, is partially determined by these activities, since the smell going from the smelling object in any other direction than the nose is obviously no longer destined to be an olfactory sensation. What one perceives as an odor is therefore partially the expression of motor acts connected with smell. The object-smell is, therefore, the expression of direct linear relationship between the smelling object and the subject. Subject Sc. after holding coffee under his nose for twenty seconds noted: "Without breathing the warm air in the nose seems to be connected directly by smell to where the object is." This relation is a particularly intimate one in psychologic experience. It probably has to do with the fact that part of the object-smell is in the nose. The object-smell is carried on a prolongation of air extending from the object to the nasal air: the smell is conveyed, so to speak, from the atmosphere of the object into the atmosphere of the nasal cavity, and there and anywhere along the course of this extension it is perceived as smell. The subject then knows of the smell, because he has perceived it outside and inside the nose. He has no immediate feeling (unless he sniffs) that he exerts any special activity in perceiving smell; there is not, in the common sense, an immediate sensation of smell.[15] The sensation of smell is objectivized, and has to do almost exclusively with the object-smell. Even though an important part of the object-smell is in the nose, it is objective, because the inside of the nose belongs physically and psychologically to the external world. The object of smell thus does not come into such close contact with the subject as, for instance, the object of touch sensation. It is remarkable that there is no directly mediated sensation of smell, whereas there is a direct sensation of touch. Smell is, like hearing and vision, an objective sense; it is much more objective than touch.

In the foregoing description it has become clear that objectivity is characteristic of the perception of smell. We realize that smell is perceived around the object, surrounding it, but we do not know from our experiments clearly what smell is. It is not essentially a sensation, but an experience belonging to the object. It is an indefinite quality of an object and as such is something objective in the outside world attached to the solid object or more or less independent of it. The term "smell" characterizes this psychologic fact rather clearly. In ordinary language one says that some one has an "air of mystery" or

15. When we say "sensation" we mean something belonging to our own body.

a proposal has an "evil smell," as, for example, in the expression, "I smell a rat."

4. Experiments with Imagination of Smell.—Our subjects were ordered to imagine the smell of a rose, of frying bacon and of burning rubber, a smell without body, as sea air or the air of a forest and the smell of urine and feces. Three of our subjects could not produce wilfully any imagination of smell. In imagining the smell of a rose subject M. said: "I had an immediate visual image which was elaborated and definite (tactile), with sensations in the nostril, but no smell." The same subject, on trying to imagine the smell of frying bacon, noted a feeling of frustration on not getting the olfactory image. Subject R in imagining the smell of a rose stated: "It was hard for me; I had a feeling of softness in the palate and a soft velvety touch on the palate, but no smell of the rose."

In general there was difficulty in the imagination of smell; optic images were almost uniformly combined with the imagined olfactory images. In some subjects the optic pictures represented the chief product of olfactory imagination, occurring as a complication or even a substitute for olfactory sensations. Frequently we have seen subjects recognize a distinct, definite "feeling" for an olfactory image without being able to say that they actually experienced an imagined smell. There is an immediate knowledge of the odor, but not a real olfactory perception. This is clearly expressed in one subject who said, concerning the imagination of frying bacon: "My mouth waters, but there is no smell, no taste and no olfactory image, only a feeling of an olfactory quality. I can't get it into my nose. Something gets within two feet of my nose and then stops." Another subject, subject P., who found olfactory imagination difficult, said: "It is largely visual, but I have the memory of a characteristic odor, though not as biting as the actual odor. If I would actually smell, the odor would be in my nostrils. It is difficult to maintain the olfactory memory. I maintain it better, but I think it obnoxious. But I can't keep the memory constant. I have to go back to the optic picture in order to find the odor. The odor is somewhere between the optic picture and me." About the odor of roses he said: "I get hold of the odor near my nose, near the tip. The image is less well defined and is more in space than actual smell sensations would be. When I breathe deeply I get the image better, but I do not feel it in the tip of my nose. When I breathe out I lose it." Subject P. got the imagined odor of urine and feces inside the nose and fairly constantly: "When I breathe deeply it goes farther up and becomes fuller, I have also the optic image right in front of me." For him the image of the smell of a forest is as if it were in his head.

Subject A., who usually had a poor imagination of smells, has a cleancut olfactory image in imagining unpleasant substances, such as burning rubber. He said: "It is a deep smell and is quite satisfying. The smell lies in space in a spiral arrangement, but I do not have an optic imagination with it. It is identifiable, but I can't smell it. I get a feeling of bodily satisfaction from it."

The various types of experience with olfactory imagination are seen in the following protocols. Another subject (B.), who was asked to imagine the smell of a rose, said: "I can't imagine it, and I get some feeling of anxiety about this. I feel that with a little help I could smell it. I know that identification would be possible if the odor would be given to me."

Subject F. said of imagining the smell of a rose: "I can't smell it but I can tell you what it's like," and of frying bacon: "I know what bacon smells like, and I feel the desire to smell, but I cannot smell it."

However, at a repetition of the experiment in imagining the smell of frying bacon, subject R said: "I imagine, visually, curled brown bacon with very

sharply pictured whitish fumes rising into the air and in the background a vague picture of a kitchen range. It is impossible to smell the bacon actually, but there is a feeling that the odor is just exterior to the nostrils and being smelled of, and I must breathe in to attempt to imagine a smell, although each inrush of air makes it harder for me to imagine the smell by changing the air in the nostrils." The same subject said: "I visualize a red rose immediately, and I have the idea of being in a perfume-laden room like a florist's shop, and I almost think I smell the perfume until I start to sniff, and then I lose the odor. There seems to be a faint odor around my face under the nose, but nothing happens inside the nostrils. Everything happens under my nose in front. When I try to carry it in, nothing happens, but I still have the feeling that whatever feeling I have is outside and external to the nose." Subject Sc. gave the following description on imagination of the smell of a rose: "There is an optic picture and a rather vivid impression of a smell which is in the nose and irradiates from the nose. The smell is also in the warm air in the nose. The imagination can't be kept for a long time. The smell is only there in the imagination as long as the air is not breathed in. The smell comes back after a while."

Another subject (S.) said, when he was asked to imagine the smell of urine: "There is a suffocating sensation in the back of the nose and the upper part of the pharynx. The odor is ammoniacal and there is the visual image of a lavatory." The imagination of the smell of feces in subject S. was: "An unpleasant but stinging sensation in the upper part and back of the nose occurs simultaneously with the fecal odor and a feeling of gastric discomfort." In general, most of our subjects have had vivid olfactory imaginations concerning these unpleasant smells; they are realistic to the point that the various unpleasant chemical components in the excretions, for instance, stand out strongly in the imagination of smell.

Sometimes even definite olfactory imagination becomes clearer when the subject does not attend directly to the subject in hand. For example, subject B. said of urine: "When not attending much to the subject in hand, i. e., to the experiments, I get the imagined odor strongly in whiffs which don't last long. These odors seem stronger then and more real than the smell coming when I concentrate attention on olfactory imagination. I see a picture of urine in a bowl and immediately I get a smell which comes in whiffs, being stronger one moment and gone the next. It is at first definitely connected with the visual picture of the bowl of urine. The ammonia component of urine is foremost. When I breathe in, the odor disappears before the onrush of air. I feel that the warm residual air in the nose is cooled by incoming air. The warm air carries the smell."

Subject Sc. said: "When I try with particular care to imagine a smell it is harder than when I imagine it in a fleeting off-hand way when the suggestion is present, i. e., when the smell has been a recent experience, when it is in the air, when it is more real than otherwise."

In all experiments, with either pleasant or unpleasant odors, we have found it simpler for most subjects to represent (to imagine) odors which they had smelled a short time before. In experiments with olfactory imagination it was surprising to find that changes in the image which we have learned to expect in optic and tactile imaginations did not take place here. When the olfactory representation does come to sensual experience it stays unchanged and soon disappears. In none of our subjects did it remain in the perceptive (sensual) field for a long time. No changes in the quality of the image and no inherent

tendencies of any kind were observed such as were described in earlier work on other sensory modalities. There is a definite relation between imagination and breathing; in a few subjects breathing destroys the imagined perception, while in others it helps.

Our results show an interesting situation regarding the localizations of olfactory imagination. They seem to occur roughly in three areas: in space, in the head and in the nose. Subject P.'s experiences demonstrate localization in the head, as when he perceived an odor "in his head" when attempting to imagine the odor of the ocean. Similarly, subject R. felt the after-effects of smell in her head: "The smell goes up from the nose diffusing on both sides into the region of the sinuses."

In subject R.'s protocol the localization was clearly set in space. On trying to imagine the odor of coffee he said: "I have sensations in my stomach, mouth and nose. I have a warm feeling in the stomach. I feel the smell on the palate and taste the smell in my nose more than in my mouth. The stomach sensation is the most important. I see brown, perceive a feeling of brown color and a dry mouth. There is a warm and light feeling in the nose and mouth. There is a sharp smell of coffee, up to the nose root. I have a feeling that now you have coffee next door—now it is in the room. The smell is carried on the air into the nose. I know it is coffee before the smell is carried in on the air. Now I feel sure that the smell is in the air in the room. At first I smelled it in me. I actually smelled it. It maintains its strength."

Subject D, on the other hand, localized the odor in the nose itself. On imagining the smell of an orange he said: "It is more on the palate than the tongue. The taste stands out more than the smell. Smell is located in the nose, not outside in the air. Smell is more in the wings of the nose. The smell when smelled, stays in one place like a heavy fog. It is like a concentrated fog coming toward the nose and pressing on and up into the nose and is smelled in the nose."

Of the three localizations, that in the nose gives a full character of reality to olfactory imagination. It is difficult to determine whether any sensual elements are connected with the olfactory picture when it is placed in the head. It is likewise difficult to define the character of the memory images when the subjects say that they know what an object smells like but cannot imagine it. We are inclined to believe that in these cases also sensual (olfactory) elements are present. From our experience in this work we do not think it justifiable to separate completely the "mere knowledge of a smell" from its sensual determinants. Henning stated that olfactory imaginations always have an eidetic character and are close to perceptions. Our protocols contradict this point of view. It is remarkable that olfactory imaginations were the more vivid the more they were related to disagreeable emotions and vegetative phenomena. Our subjects could generally imagine the smells of feces and urine more easily and with greater intensity than any other smell. In the one subject who experienced the smell of burned rubber so graphically, agreeable sexual feelings were connected with this odor.

He said: "I have a feeling of bodily satisfaction—I often have feeling akin to sexual satisfaction with the smell of rubber. I seek it out to smell it."

5. Taste: After-Effect and Imagination of Taste.—For comparison we conducted a few experiments on the imagination of the gustatory sensation. We first tried to study the phenomenology of taste. The sensation of taste that is ordinarily present in the mouth in the absence of food seems to exist on the lateral and posterior part of the tongue, extending toward the pharynx. It is partly a tactile sensation and only in part an actual taste. It is difficult to say how much is the characteristic taste perception. Certainly motor elements and salivation are intimately connected with taste sensations of any type. The teeth appear to be related to taste sensation, possibly as a sort of reservoir from which taste sensations are derived in the absence of food. Thus subject B. said: "There is taste on the sides and back of the tongue. It seems to come off the teeth; it goes down to the pharynx and toward the stomach. It is difficult to be attentive to the taste sensation without swallowing."

None of our subjects had any difficulty in imagining taste. In studying the imagination of taste we asked the subjects to imagine taste without any particular food or substance. It was possible for our subjects to imagine a spontaneous taste in the mouth, but with it were clear optic images: a chewed and tasted object or the tongue was always present in the accompanying optic imagination. The optic image accompanied the gustatory image almost necessarily. But if one imagines an object outside the mouth and at the same time one imagines a taste in the mouth, it is apparent that there is no connection between this external object and that which is chewed, swallowed and tasted in imagination in the mouth. In other words, the imagination of taste is related to that which is brought into and tasted of in the oral cavity. In imagining sweet taste subject Sc. said: "I have a vivid imagination of taste, but the taste is in connection with the feeling that I drank some sugar water. I am conscious of the saliva and some tendency to swallow. There is a sensation of sweetness in the lower part of the tongue and near the root of the tongue." On imagining a bitter taste he said: "The imagination is less vivid, more toward the posterior part of tongue with the motor reactions of gagging."

The motor elements of gustatory imagination are strong; one feels the chewing and is keenly aware of sensations of swallowing, the movements of the tongue and tactile sensations connected with the buccal mucous membranes, teeth, pharynx, etc., and optic pictures involving these elements. On imagining eating meat subject Sch. said: "I get a rather vivid image but again the taste is toward the root of the tongue with a tendency to swallow. There is also some sensation of chewing and moving of the tongue. The motor part is strong. The taste is imagined as a succession of acts of swallowing increasing with every imagined act of swallowing. Many tactile sensations are connected with it."

The results in gustatory imagination were uniform in the subjects. When an edible object was imagined, it was imagined visually outside the mouth and also inside the mouth. The taste, however, was connected only with the object in the mouth. There is a distinct difference between the tasted object and the sensation on one's tongue and mucous membrane.

The following protocols show the relationship of the tasted object to the sensation of taste: Subject Sc. imagined tasting cheese: "I have a vivid optic picture of the cheese. The smell is around the cheese and goes into the nose. There is no taste on the cheese, but the taste is in my mouth in the same way as if

I imagined eating the cheese." Subject B. said: "I visualized the cheese behind the glass partition in a store and I had fleeting tastes of it, but my imagination of taste increased in vividness later. There was not much smell. The taste was certainly in the mouth in the posterior part. It had little to do with the cheese in the cabinet."

It is evident from these protocols that the imagination of taste is a taste perception rather than a taste sensation in the sense already stated. In the imagination of taste as well as in the after-effects of taste, strong salivatory and motor impulses are intimately connected with the perception.

OLFACTORY HALLUCINATIONS: CASUISTIC MATERIAL

A search of psychiatric literature fails to reveal any psychologic studies concerning olfactory hallucinations, especially among psychotic persons. Brill emphasized the lack of material on this score, but indicated the work of the analytic school, in which the rôle of smell in the development of neurosis and in the unfolding of the libido has been studied (Freud, Ferenczi, Jones). Textbooks practically neglect the subject. Bleuler [16] gave passing mention to olfactory hallucinations in schizophrenia; other authors included it among "other types" when discussing hallucinations. We have considered it valuable to study olfactory hallucinations, especially with the results of our experimental work in mind. The cases to be presented were picked serially from clinical cases, the only criterion being the presence of olfactory phenomena. In a relatively short time we have examined forty cases from the clinical material at the Bellevue Psychiatric Hospital. These fall in the schizophrenic and alcoholic groups chiefly; others present conditions of organic origin—cerebral arteriosclerosis, senility, epilepsy—and also psychoneurosis and neurotic homosexuality. Only the pertinent portions of the case records will be given.

Case 1.—Adelaid V., aged 55, a Negress, in whose case the clinical diagnosis was schizophrenia, paranoid type, said: "Fumes are administered under my door and through the radiator pipes. I hear them saying that they are going to kill me. Five years ago a woman put chemicals under my clothes. Some of the smells go right through you, you inhale it, it goes into your bowels. Then when you go to the toilet it has a terrible odor. It is very disagreeable. Some odors come through the window and smell like acid. Sometimes the gas is odorless. Another smell is like gas. The odor comes out of me when I breathe deeply. I have to breathe deep to get it. Sometimes it smells like sulphur; the odor is suffocating. The gas rips through the stomach."

Case 2.—N. S., a woman, aged 35, with schizophrenia, paranoid type, said: "My husband put opium in everything. It smells like dead bodies; it smells like

16. Bleuler, E.: Textbook of Psychiatry, New York, The Macmillan Company, 1924, pp. 64 and 342.

rotten meat and the toilet. The whole room smells. My husband smells on his feet. Bad women put poison in the milk. I have to run to the toilet three or four times."

CASE 3.—Bridget H., aged 69, with schizophrenia, paranoid type, said, while rambling: "It is a bad smell—it is of the clothes. The odor comes to me when I hang clothes in the closet. It is like meat which is too long in the box. My daughter-in-law has a bad smell. It is a dead smell. I am so clean that I often have to take three baths a day."

CASE 4.—Diana M., aged 33, with schizophrenia, paranoid type, said: "I smell dead spirits. It is like rotten meat; it is all over the house. (Is it necessary to sniff?) No. As soon as the spirit leaves the body it smells. My husband smells bad when he runs around with women. But it is a very different smell—a natural smell."

CASE 5.—Grace M., aged 50, with schizophrenia, paranoid type, said: "A stable-hay smell is put on me. Especially on my hair. Other people smell it. At first I thought the smell may come out of the body; it may be halitosis or carcinoma. It was terrifying. Later on I thought it came off my hair." She has always been sensitive toward smells.

CASE 6.—André H., aged 50, with schizophrenia, paranoid type, said: "A smell of dirty cabbage came directly to my nose. Sometimes I thought it may come from my hair. I always loved flowers. I prefer flowers which smell."

CASE 7.—Helen C., aged 42, with schizophrenia, paranoid type, whose remarks were rather disconnected, said: "I detected a bad smell sixteen or seventeen years ago. It was overcoming; it affected the heart. It was like cigaret smoke. It was on the clothes, as if somebody would throw something at you. The smell is always of the same type. The smell is in the nostrils. I have always been sensitive to smell. When I was at school as a child and something was wrong at home, I could tell—I got a nervous feeling and the presentiment of smell was connected with it."

CASE 8.—Cecelia B., aged 40, with schizophrenia, paranoid type, complained about a nasty musty smell like dead mice. She had a vivid memory of smells, especially of her baby when it was small.

CASE 9.—Erma M., aged 55, with schizophrenia, paranoid type, said that she smelled gas and dope. "It is choking and more disagreeable than the smell of the toilet. It was in the room. My smelling is very good. I felt gas inside of me. I was half asleep."

CASE 10.—Louisa H., aged 43, with schizophrenia, paranoid type, complained that "they would send stenches up—stool, urine, vomiting."

CASE 11.—L. B., aged 40, with schizophrenia, paranoid type, complained that the father is haunting every orifice of her body. He comes in and out of each orifice at will and makes a terrible smell like gas or like ether. He makes green stuff run out of her.

CASE 12.—Helen M., aged 68, with schizophrenia, paranoid type, claimed that she was a florist, and had an exact memory of all kinds of smells, especially of roses. She complained that ether was coming from the apartment below her. People persecuted her. She had been operated on for an extra-uterine pregnancy many years before and remembered the smell of ether.

CASE 13.—Charles T., aged 35, with schizophrenia, paranoid type, who was disconnected, felt that his father and brother exuded an odor when they had

sexual intercourse. He called that a "trance." When he felt the strong odor he heard the word "rose." It was like a vapor coming out of the system. This vapor would put everybody to sleep, but did not put him to sleep completely. It was an odor coming out of the male and female systems which accumulated in the room.

CASE 14.—Constance P., aged 33, with schizophrenia, paranoid type, heard voices of male and female persons. "I hardly remember the words. When I hear the voices a whiff of offensive odor like stale urine, bilge water or water from the sewer comes right to my nostrils."

CASE 15.—William F., aged 43, with schizophrenia, paranoid type, complained about the "heavy" smell which he smelled without sniffing.

CASE 16.—Mary W., aged 53, with schizophrenia, paranoid type, showed considerable deterioration of intellectual life; she had a diffuse and all-inclusive delusional system, with numerous olfactory hallucinations. She was incoherent and showed signs of advanced schizophrenic dilapidation. The picture can best be visualized by the following statements: "Quite a number of years ago there was a Saturday infection. I told a detective and he told me they were probably making beer. It was a terrible odor. I didn't think of an infection then. I thought it was from the basement. I did not think they were trying to do anything to me. The first gas is very strong and unexpected. When I open the doorway I'm caught suddenly. The smelling and feeling all come at the same time. The second gas smells like burning hair. It's in the whole room. It has been coming from the roof. The decomposing bodies (odor) came through the corridor. It comes through the opening in the toilet. Sometimes it comes from the kitchen, from the floor. (Do you think your body smells?) No. (Do you think about it?) Sure. After the gases had been in they got disengaged from my body—from the nose, the rectum, the clothes. (Is the smell different from that of your body?) This has nothing to do with the smell of my body. I swear it—I could take a solemn oath on it (becomes irritable). (Do people say that you smell?) No. I shall tell you the secret. I have written a thesis—'Fantastic Elements.' One must never tell the title, but I tell you. Do you want me to swear to secrecy?"

A table prepared by this patient resembles a paradigm of all possible hallucinated smells. As such it is of interest from a psychopathologic point of view.

CASE 17.—Daisy M., colored, aged 42, with schizophrenia, paranoid type, had numerous ideas of somatic change. She hallucinated actively, became emotional, and was depressed at times. Probably the case was one of schizophrenia, but the question of melancholia with hypochondriacal content was entertained. She complained about an odor coming from her stomach. She said that everybody notices it. It must be a tall worm. The doctor says it is because she did not menstruate for two months. In the last three weeks her nose and mouth have got bigger! She has become ugly. She was good-looking before. The patient became agitated when speaking of the somatic changes. She wanted to be cut up, and said: "I think if they cut me up and see what's inside they can tell." She smells like manure. It comes from the bowels. "The smell comes up to my nose and causes my nose to bleed. Stanley told me that I smell and that a worm comes out of me. He is drawing my whole inside down. He made me take douches day and night. My smell troubles the neighbors."

CASE 18.—Louis D., aged 39, with paranoid schizophrenia, complained that he was gassed from the next building. He felt doped and weakened.

CASE 19.—Rachel H., aged 29, with paranoid schizophrenia, said: "I think because of my ear trouble I have a bad odor. They said I smelled bad; that I don't take a bath. I never associated with anybody on account of the odor. I smell the odor myself. The smell comes from the ear."

CASE 20.—Dorothy T., aged 39, with paranoid schizophrenia, complained that she was molested by bad smells which come through radio wires. It is like dog refuse or sweat. It is a filthy, foul smell.

Hallucinated Smells Listed by Patient 16

	I	II	III	IV	V
Odor	Sharp, acute	Dull; mucous; opium-like	Decomposing bodies; foul	Disinfecting (gasoline)	Noxious fumes
Duration	Very transient; condensation type	40 minutes	Indefinite	About an hour	
Organs affected	Lungs: breath; bowels: swelling, bloody exudation; stunning; faint and dizzy	Digestive system: fermentation; narcotizing	Stomach: vomiting; nausea; oppressed feeling over chest; perspiration	Irritating to tissue	Makes bowels move
Mode of administration	Thrown into path (meaning ?)	Put into closets and other closed places	By means of hammering between the ceiling and wall; putting it in these openings	Gasoline tank on fire escape on floor below; through halls, windows and floor	In James' kitchen
Time administered	Unexpectedly—only at night when awake; later changes it to daytime when she goes outdoors	Daytime when appointments are made for lessons; on Saturday evening especially	On Saturday evening especially; on particular days of appointments	Almost constantly; six times a day	Saturday evening and one Sunday afternoon
By whom administered	Eighteen apartment neighbors; they put it outside the door	A woman who poses as her student—Mrs. Holmes, 275 Central Park West; she pretends to take lessons in Italian but hypnotizes patient; goes into trances; discharges sparks through her nose; also Mrs. Mahoney, Apt. 15	A detective or a gangster posing as a plumber	Called "Crimens," lives in apartment below; may be political or antipolitical, etc.; acts in harmony with police	James, a very nice preson

CASE 21.—Hugh H., aged 58, with alcoholism in an arteriosclerotic psychosis, said: "People notice my smell. They start to move away. I have an idea that they remark that I am a fairy, and throw things at me. I started to notice it about March. There is an awful odor coming off me. They say that it is an awful terrible stench—like dropping urine inside my underwear. I heard somebody say, 'There is pansy here,' and the next thing I smelled an odor in back." He insisted that the odor comes from the back. "I have taken baths every night and changed my clothes, and the next minute I have the same odor. People in the subway couldn't stand next to me. It smells like citronella. I heard a detective say that I was the dirtiest fellow in New York. . . . My penis is small and erections are not so good since my nervous breakdown. I never did any fairy business in my life. I don't think it is the natural thing to do. The odor is noticed by men and women. I lose semen. It gets around the clothes."

CASE 22.—Adolph B., aged 43, with an alcoholic psychosis, acute hallucinosis, who was apprehensive, delusional and excited, said: "I was told to go to New York to report in. The message came on the radio—it was from the President and they said I was still a soldier and had to be shot at sunrise for desertion. It is a small wave and I hear it in my ears. They say I'm rotten, syphilitic and everything. People say I stink on the street—I never smelled it. They keep talking about me being a bum. Some one wanted to shoot me. The President wanted me operated on—cut up in pieces. I heard that my stomach was rotted out. They could tell by the smell. I heard them talking about me. They said, 'he is rotten—get him out of here—he hasn't got a chance in the world.' I thought I smelled; I got so ashamed I wouldn't walk on the sidewalk. It smelled rotten, like a rotten body. I smelled a man being pulled out of the water once—it smelled like that."

A sketchy account of the personality from an olfactory point of view is of interest: "I started sex relations at 16—regular until four years ago. Quit then on account of fear of disease. Always was particular about washing myself with different solutions; was sensitive toward odors as a boy. (Are you sensitive to smells?) Not that I know of. When I was little I used to get upset by a smell. One time I picked up a can that smelled rotten—there was rotten fish in it and I smelled it for a week. I can never stand the smell of rotten clothes standing around wet. I was around 5 or 6 years old when I smelled that can. I wash my hands very often. I noticed that in a public toilet I couldn't stand the smell of another person. I never paid much attention to feces, etc., or to jokes about them. I would rather hear a smutty joke about women than about fecal matter. I never liked the smell of women—particularly at the time of the period—it was a kind of sickening affair. I like the smell of cooking and flowers—I love flowers. But I like the smell of perfume."

CASE 23.—Charles D., aged 35, with alcoholic hallucinosis, complained that gas in the room puts him to sleep: "I don't know what they did to me. They tell me what I am thinking about. They played every song I suggested. Some vapor gas was blown in like peroxide. It puts me to sleep."

CASE 24.—Thomas H., aged 38, with alcoholic hallucinosis, complained about the urinary smell akin to sulphur put on him by his brother.

CASE 25.—Charles W., aged 42, with alcoholic hallucinosis, who had been a constant drinker for years, said: "Some men wanted to come into my room and lay pipes under my bed to fumigate the room. They followed me around the room with special lights. I smelled some of the fumigating liquid from the apartment above. Some of it leaked through. It has a very strong smell, burning your nose, bringing tears to your eyes. When the liquid drops on you it burns and goes through the pores and through your system.

"It had a smell like black ink—some ink is stronger than others—especially black ink. . . . No matter how clean you keep a toilet, there always is some smell. When manure gets old, it liberates ammonia and catches you in the nose. Some women have disagreeable odors, especially about their sex organs and armpits. You always get some odor. It is very disgusting to me. I have thought about such things a great deal—I prefer them without smells. I like perfume, but some perfume is disgusting, even more than urine."

CASE 26.—Herman V., aged 63, with alcoholic hallucinosis, who gave a history of chronic alcoholism, smelled gas around the room: "It was like the smell of chloroform or gas."

CASE 27.—Ernest H., a Negro, aged 36, with alcoholic hallucinosis, complained about the smell of feces in connection with a complicated homosexual persecutory picture. He had hallucinations in all spheres: delusions that gas was pumped into his body and was pulling his flesh. He gave a history of chronic alcoholism with recent heavy drinking. He said: "A woman was spraying the room with gas, like you kill roaches. It has a sharp choking feeling. It got full of it. Smells like chloroform. Then the room got foggy. It got on my skin and it draws the flesh up." He said that the smell was like formaldehyde. He knew the smell of paraldehyde from his work at an undertaker's parlor. (Do the smells annoy you?) "No, I could eat my lunch right here with a body five weeks old next to me. I remember down in Florida two kids killed an old man. They dug a little hole and put him in it. He smelled terrible. (Do you object to the smell of women?) Not of some of the women. Some of them smell rotten. (Is the smell of a toilet objectionable?) I don't like it, but it don't do me any harm."

CASE 28.—Elizabeth B., aged 28, suffering from a carcinoma of the uterus which actually gave off an offensive odor, felt very sorry about it. She had the idea that all people realized it and made remarks about it.

CASE 29.—Mamie P., colored, aged 40, with alcoholic hallucinosis, was confused and tended to be rambling and irrelevant; she was tense and a little evasive. She said: "I was drinking a lot of wine and anisette and I got noisy and it made me vomit. I thought I had worms. I felt them in my stomach and in my rectum. I got some worm pills. After I took them, they started up my throat and almost choked me to death. I opened my mouth and I thought I saw one and I went to screaming and hollering. I can taste a sweetness when they come up; sometimes it smells like stool. I know I'm full of worms. When I was drinking I was smelling things—it stunk like chicken-dodo (feces). It smelled bad; not like a dead something rotten, but a different stink."

CASE 30.—Joseph McH., aged 45, with alcoholic hallucinosis, said: "They were shooting at my throat. It hit me and struck a fire and the fumes came up my nose—I was supposed to be put on the spot because I tipped off my friend. It hit me ten shots. Then it went into my nose. It was a kind of gas smell—it had a little stink to it—something like gas from gas-works."

CASE 31.—John Q., aged 72, with senility with moderate deterioration, complained about the smell of ammonia and compound solution of cresol all around him. He said that one uses ammonia for the toilet. Ammonia smells like stale urine. He told with gusto a story that he saw in Ireland a factory where they used to wash flannel in urine in order to make the quality of the flannel better.

CASE 32.—Dewey R., aged 30, with alcoholic hallucinosis with schizoid features, was apprehensive and talked at length about his delusions and hallucinations. His expression was a little stilted; he was preoccupied with his thoughts. He said: "These two fellows lived underneath my apartment. They put electric on the floor and attached it to the bed. They burned me and I could smell burning flesh. It came from all over me. Everybody on the outside keeps shouting, 'Take him out, kill him.' A daughter of this man would shoot poison pens at me. It made me groggy for a while. A chloroform odor came from the pens. They blew it in the back of my arms. I just can't stand the smell any longer."

CASE 33.—A man, aged 35, experienced an epileptic aura of a smell like burnt flesh.

CASE 34.—A man, aged 24, who was acromegalic, following a cranial operation experienced epileptic fits with an aura of smell. The smell was like wet laundry and like that of a chicken coop.

CASE 35.—Myrtle S., aged 25, colored, probably with vascular syphilis, presented a manic picture with toxic features. She saw a bright light, smelled flowers, saw people who had been dead for years, saw God and heard voices.

CASE 36.—Alfred N., aged 19½, of a neurotic type, complained about an odor coming from his mouth after he has been kidded by friends. He had a hypochondriacal attitude toward his whole body. He was very neat.

CASE 37.—Stephen R., aged 26, with a neurosis or beginning schizophrenia, had attacks in which he felt that he exuded a strong odor from his body.

CASE 38.—Caspar F., aged 22, a homosexual prostitute, with a compulsive and obsessive neurosis, complained about an odor which exuded from his body and his bowels like swamp. It was different from fecal odor. He said: "For the past three months I've been giving off a body odor. It smells like a swamp, dug up mud, a rotten smell; it comes from my guts. I can't go anywhere. I don't want to go with my friends, and I think I've lost them on account of that. It doesn't stay with me all the time but comes on three or four times a day. That's the only thing that bothers me. I feel fine. I don't go to girls for that reason. I smell it through my nose. I smell it all over the body. I think it stays 6 feet around me. It smells more in front of me. Sometimes it smells like a swamp. I never experienced something similar. When I go to move my bowels, the smell is different. I was not sensitive to smells as a child. (What do girls say about the smell?) I was with a lady (35 years old), and when the smell came on she walked away. I wanted to have relations with this woman. I met her in Central Park. I think this smell comes from my stomach, because after I eat, I smell the smell. It happens sometimes the odor comes and I masturbate."

CASE 39.—A woman, aged 45, with an atypical depression, had hallucinations about animals which were around; she smelled their urine and excreta, smelled this in her food and in the bathtub.[17]

CASE 40.—Alphonsus L., aged 25, with schizophrenia, hebephrenic type, was tense and had numerous hypochondriacal delusions and misinterpretations. He had ideas of reference, with auditory hallucinations, feelings of guilt, of sin and of unworthiness. He was somewhat apprehensive and uneasy. He said: "The orderly said something about the odor going all over the ward. When I sweat the odor comes out. It smells like s..., but sometimes it smells like decayed animals—like rotten things. The odor clings to the body. Even when I don't have a bowel movement it comes out in the sweat which smells the same. The smell comes up from my crotch along my stomach and chest and I smell it. It stays in the atmosphere around me. It always is the same—no stronger or weaker.

"When I was a kid about 7 or 8 years old a fellow fooled around with me and I got a stricture from that; when I was 11 my brother had intercourse with me. I was treated up in Medical Center for it until lately. I am kind of sensitive. I am afraid of offending people. In the church one would tell me I had to do so much penance and the other would tell me I didn't have to. I have

17. Schilder, P.: Studies on Depressive Psychosis J. Nerv. & Ment. Dis., to be published.

been sick over a year suffering from this stricture. When I'd go to the toilet I couldn't get rid of the smell. If I walked, that odor came up and I would be able to smell it five hours later. The boarders noticed it. It was worse than a body odor—it was from the rectum. If I went into a show or into church, they noticed it. I was asked to get out of the theater. They would sniff—just looking at me."

COMMENT

Even a casual survey of the material reported will impress one with the frequency with which smells connected with the body occur in hallucinations of this type. Careful analysis bears out the fact that the smell is of something organic, such as is found in decayed substances or feces. It is opium, which smells like dead bodies, or it is like meat which has been too long in the box. Dead spirits smell like rotten meat. There are smells like halitosis and carcinoma; smells of decayed cabbage; stenches like stool, urine or vomiting; musty smells like dead mice and a terrible smell like gas, or like ether. A strong odor connected with sexual intercourse is noted. There are smells like stale offensive urine and water coming from the sewer or like bilge water; smells like rotten egg or like decomposing foul bodies; odors coming from the stomach and from the genitals, or sweat odors, like urine inside the underwear; bad odors coming from the ear, or from the back; a smell like refuse of dogs or chickens; a urinary smell akin to sulphur; smells coming from a carcinomatous uterus; a smell like ammonia and compound solution of cresol, comparable to urine; an odor coming from the mouth; attacks of odor coming from the body; odors of wet laundry; a swamplike odor coming from the body and an odor of burned flesh.

Bleuler noted that hallucinations of smell are often of "disgusting and poisonous substances . . . including pitch and sulphur." In a discussion of parosmias (perversion of smell perception) due to organic causes, Seydell [18] remarked that these smells "usually have a disagreeable odor," whether due to local or to cerebral causes. He quoted the case of a priest to whom all odors resembled cabbage or manure (case 6). In our experimental results it is interesting that the subjects could imagine the odor of disagreeable substances more vividly than pleasant smells. In our whole material there were only six instances in which the smells which have been hallucinated had nothing to do with odors either from the patient's or some one else's body. In three cases it was a smell like ether or chloroform. In one case, with a strong organic tinge, there were agreeable smell experiences.

In many patients the smells have a suffocating or narcotizing influence. It is a common notion in psychopathology that a smell has an influence on the state of consciousness. A smell often conveys with

18. Seydell, E.: Olfactory Disturbances, J. A. M. A. **99**:627 (Aug. 20) 1932.

it a change in consciousness. In common speech one hears the phrase: "The smell was so strong it almost knocked me down." The most complete description was given by the patient in case 16, who in some way summarized all possible disagreeable smells. There is a deep psychologic relationship between the two main characteristics of hallucinated smell. The foul odor (the odor of decay) is at the same time the odor that takes away consciousness. The patient in case 2 said: "My husband put opium in everything. It smells like dead bodies." In case 13 the odor exuded by the father and brother during intercourse puts the patient into a trance. In case 11, the spirits which are haunting the inside of the patient smell like death and like ether. Daly and White spoke about the hypnotic power of the sex-attracting odor given off by the female in heat. In this there is a suggestion that may explain why so many patients complain of fancied attempts at narcosis in connection with the hallucinated odors. In case 41, a woman with gonorrheal arthritis had a delirious hallucinatory episode in the early stages of the infection in which she said that there were smells like dope or ether in the room. The next morning she awakened with a vaginal discharge. She felt that she had been sexually abused by a neighbor who had also put the ether-like smell in her room.

In the majority of our cases the smell came from outside the body, although it was still recognized as a body smell. Some patients complained that the smell was put on and into their bodies and was then sent forth from them as their own odor. Characteristic examples of this are the statements in case 5 ("A stable-hay smell is put on me, especially on my hair. Other people smell it. At first I thought this smell may come out of the body. It may be halitosis and carcinoma.") and case 6 ("A smell of dirty cabbage came directly to my nose. Sometimes I thought it may come from my hair.") The clothes often carry the smell as an intermediary object. In case 11 evil-smelling spirits were in the body. The patients in cases 22, 28, 29 and 36 complained merely about the smell coming from their own bodies. In case 28, it was merely an exaggeration of an actual smell connected with the body. Although all smells coming from the various orifices of the body are of importance in the hallucination, the anal smells are, according to our material, of special significance. But even with other odors there is still to be found the general idea of decay.

The olfactory hallucinations of the patients with alcoholic hallucinations are not fundamentally different from those in patients with paranoid schizophrenia. The latter form the largest group in our material. Brill also emphasized the frequency and poor prognostic indication of olfactory hallucinations in patients with schizophrenia. Bleuler stated that olfactory hallucinations probably "belong to a fundamental schizophrenia." It is common to hear in neurotic patients, especially those

with severe obsessional neuroses who may be close to schizophrenia, complaints of the odors of their own body occupying the foreground of the clinical picture. Brill noted, in discussing fetishism in neurotic patients he had analyzed, that the smell of the dead, anal smells and preoccupation with odors have close interrelations with each other. In a case analyzed by one of us (P.S.) the relation between feces and decay and death was obvious. The association between homosexuality, perfumes and interest in "anality" is expressed in our cases by the fact that many of those with hallucinations had outspoken or overt homosexual characteristics. The literature of the period of decadence in the nineteenth century furnishes abundant evidence for this relationship, as in Huysmans' "A Rebours."

It is difficult to account for this attitude toward disagreeable odors on the part of human beings. It is probable that it concerns a primitive instinct to avoid decay and death. Lévy-Bruhl,[19] in his work on primitive mentality, reported the primitive attitude toward death. He related that the primitive mind does not regard a person as dead until obvious signs of decomposition appear in the body. Among some tribes the funeral ritual does not commence until decay is in evidence; until then the corpse is placed in the council circle, is brought food, etc. When the body is decayed it is represented as an evil spirit, from which the good spirit (the soul) has departed. The odor is an important sign of decay. There are certainly primitive respiratory reflexes which protect against this olfactory danger. There must be other primitive reflexes and primitive instincts which protect against food that has gone too far into the decayed state. Many of our patients complained about biting smoke of suffocating character. The odor of the dead is often described as penetrating. Our material does not help in the final analysis to determine how these primitive defenses blend with the pleasure derived from anal smells. Another possibility would be that during the higher and later development of the personality the primitive pleasure of odor and anality was denied and repressed. It is to be considered also whether the self-protective mechanisms of the ego are not a factor in helping the strong repression of anal impressions. What is anal is decayed, and decay is equated with danger and death. Investigations like ours bring into the foreground of attention the sensory experiences which are generally hidden from consciousness. We do not deny, of course, that smell has other important functions on a more obvious or conscious level. For instance, it provides orientation concerning food or, expressed more primitively, concerning prey, and in this way is in close connection with the function of taste.

19. Lévy-Bruhl, L.: How Natives Think, New York, Alfred A. Knopf, 1926. p. 301.

Thus, the sense of smell seems to have several well defined functions. It provides a means of orientation in the external world. Tilney [20] pointed to the tremendous meaning smell has acquired for Helen Keller in her contact with the outside world. Although her sensory organization for tactile and olfactory functions was that of the average person, her "sensory supremacy (in these senses) lay entirely in the realm of intellect." It was the extraordinary richness of "associations" for olfactory stimuli that made this mode of perception so meaningful for her. For the average person there is no need for development of this particular aspect of smell, and the importance of smell in his psychic structure has consequently diminished.

But, as already stated, these functions of smell are acceptable to the ego since they deal with orientation concerning food and space generally. On a more primitive level smell has the function of protecting from obnoxious influences in breathing, of warning against decay in every form, of helping to get anal satisfaction and in conducting to a heterosexual love object. In the hallucinations of psychotic patients, also, all these functions are represented.

Our experimental findings show how smell serves as a means of spatial orientation. Smell is essentially an objective sense, quite unlike tactile sensation, but similar to vision and hearing. The fact that there is no real subjective part to the sensation of smell means that human beings have a direct means of informing themselves as to their relation to the environment of smell; they can also relate the smell to the environment, to wit, they can move away. As a psychic identity one does not come therefore into more than an indirect contact with the smelling object. The object is always external, i.e., belonging to the external world. Smell does not occur in the mucous membrane of the nose, but occurs in the air in the outside world and then goes to the air lying in the nasal cavity enveloping the mucous membrane. The mucous membrane is part of oneself; the air in the nasal cavity serves one, but still, in the inner perception of the model of the body, is not really part of one. In this way, if one smells something in the nasal air it is not reckoned as a subjective act, but as an objective act. It is an objective act of the sense organs of the body acting on a closely affiliated yet independent substance, i.e., the air. In taste, also, the object which is tasted is external to the soma, by which is meant the tongue, mucous membrane, etc. Taste is on the object in the mouth. There is at the same time an external visual picture of the tasted object, which itself is surrounded by taste. In taste, then, there is a less outstanding objectivization of the sense than in smell, but it is equally definite.

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20. Tilney, F.: A Comparative Sensory Analysis of Helen Keller and Laura Bridgman, Arch. Neurol. & Psychiat. **21**:1227 and 1237 (June) 1929.

From a social point of view this objectivity of smell has a meaning in interesting people in their own body odor and that of others. A whole fabric of social taboos and customs has developed around the idea of the objective importance of the smell of one's own body. The basis of social forms that have ruled the world for centuries—aristocracy for instance—has been rooted at some point in the varying reactions toward the objectivity of smell. The smell of one's own body also has importance psychologically, in that it gives one sense data, and it has a special significance in that it orients one about the degree of decay going on in the body. From this standpoint the value of smell to the body is threefold: First, it is an indicator of health or disease; second, it serves as an erotic stimulus, and third, as a social guide. The interrelation of these functions of smell are clearly seen in the analyses of neurotic patients. In a case analyzed by one of us, a special disgust concerning feces and decay and death followed a rather active early period of anal sexuality. The same patient complained about the ugliness of his body and of its slight erotic value. He had a tendency to estimate female beauty in an exaggerated way. When a person becomes interested in the smell of his body, he regards his body like any other object. The smell of his body remains in this respect an objective smell. Von Skramlik justly emphasized that children have a different social evaluation for bad odors than do adults. It was as unexpected as astonishing that in the great majority of our cases agreeable smells did not appear in the hallucinations, in spite of the fact that some patients emphasized their keen sense of smell and appreciation of agreeable odors.

The experimental results have given interesting material with regard to the meaning of olfactory experiences to the perceptive sphere. Olfactory experiences occur in varying intensity in various persons. Henning [21] had the opinion that one perceives in smell an intuitive picture, a "presented" picture (*anschauliche Bild*), which is a primary sense picture that goes beyond the usual type of experience. He thought that one cannot differentiate in smell between perception, memory images, after-effects, eidetic pictures and imagination and fantasy pictures, but that one experiences an *Urbild*, a primary picture, which precedes and usually replaces such smell phenomena as memory images, eidetic pictures, imagination, etc. From our experiments we do not think that smell is in this respect different from other senses. We find that perception, eidetic images and imagination are differentiated as they are in other senses, even though the experiences themselves may be vague.

In the actual phenomenology of smell it is remarkable that there is no difference in quality between olfactory hallucinations and actual

21. Henning, Hans: Das Urbild, Ztschr. f. Psychol. **94**:273, 1924.

smells. Patients describe the act of smelling and breathing (sniffing) associated with smelling in hallucinations exactly as our experimental subjects did in real perceptions. If one doubts (as did Kronfeld and Karl Schneider) that hallucinations have real perceptive qualities one should study hallucinations of smell. Nor does the factor of organic etiology of the psychosis have any bearing on the vividness of the smell experience (hallucination). In other senses, as in touch, a different situation obtains. Bromberg [5] showed that tactile experiences in patients with an alcoholic psychosis have more activity and more vivid perceptive qualities than in patients with schizophrenia. In schizophrenic and alcoholic patients olfactory hallucinations have alike the character of real and immediate perception.

As we have observed in patients with functional mental disease, the outstanding subjective characteristic of the hallucinated odor is its disagreeableness. It is striking that in hallucinations or illusions in organic conditions the same characteristic is noted. The conditions in which these phenomena occur are epilepsy with aura and tumors of the brain, especially of the temporsphenoidal lobes or rhinencephalon. Kennedy,[22] in his discussion of temporosphenoidal tumors, gave an excellent description of these so-called uncinate phenomena. Hughlings Jackson described, in 1876, a type of fit associated with taste and smell experiences due to irritation of the temporosphenoidal region, and called this phenomenon "the uncinate fit." Kennedy gave numerous examples of the "fit" from his own material and that of others, which are instructive in this study. He quoted the case of a patient who, following a head injury, suffered seizures preceded by the experiencing of a disagreeable odor "sometimes of smoke and sometimes of fetid character." Another of his patients described a vision of a "strange bad woman clad in rags" and experienced at the same time a "most evil odor," which the patient felt originated from the strange woman. A third patient described "a smell in the ward, around and about me (never in the nostrils), neither pleasant nor unpleasant, something like a factory or brewery." The fourth patient said that before an epileptic seizure he tasted "something like ink" and smelled an odor which he never described. Another patient described the smell which preceded a convulsive seizure as like that of a "foul pipe" or like "coarse tobacco." Another described a "horrid smell of rotten fish." During this aura the patient would suddenly spring up in alarm and with gestures of repulsion and disgust exclaim, "Oh, the smell!" This was followed by a generalized convulsive seizure. Kennedy [23] gave an example in a case of temporosphenoidal

22. Kennedy, Foster: The Symptomatology of Temporosphenoidal Tumors, Arch. Int. Med. **8**:317 (Sept.) 1911.

23. Kennedy, Foster: Tumors of the Intracranial Cavity, in Nelson Loose-Leaf Living Medicine, New York, Thomas Nelson & Sons, 1929, vol. 6, p. 119.

tumor in which the patient described a sensation of "a foul odor," "an awful smell of rotten weeds," which preceded a convulsive seizure. In a case of epilepsy cited by Marcus [24] in which an organic lesion was found in the uncus, the patient spoke of a "horrifying" smell, and Kutzinsky's [25] patient complained of the odor of a "stink bomb." An epileptic patient under our observation complained of smelling "rotting meat" during his aura. In other spheres in which an organic condition causes hallucinations, such as the optic sphere, the hallucinations have a less objective character. The olfactory hallucinations are completely objective, alike for organic, toxic and so-called functional diseases; objectivity is an inherent quality in the perception of smell.

SUMMARY

I. Twelve normal persons were studied to ascertain their subjective reactions to olfactory imagination.

Our purpose was to study the various categories of the perception of smell (images, after-effects, memory images, imagination, etc.) in order to understand the problem of olfactory hallucination.

During the experiments we tested:

1. Sensations experienced when not breathing.
2. Sensations on breathing.
3. Perceptions when smelling odorous objects (including after-effects).
4. Experiences when imagining odors.
5. Sensations of taste with nothing in the mouth and with food in the mouth, (including the after-effects).
6. Experiences when imagining the taste of food.

II. Experimentation concerning the phenomenology of smell showed that:

1. Smell is essentially objective in its localization; it comes "off" the object. The odor is perceived as coming from the reservoir of air in the nose. From the object to the nose is a continuous stream of smell which is perceived finally in the nose as the object itself. One can speak of this, then, as the object-smell.
2. Smell is carried in on the in-coming stream of air.
3. Smell is perceived, not on the mucous membrane, but in the warm envelop of air coating the mucous membrane.

24. Marcus, quoted by Boernstein.[10]

25. Kutzinsky, quoted by Seydell.[16]

4. There must be a motor factor in smell consisting of (*a*) a movement of air into the nose and (*b*) movements of smelling, such as sniffing reflexes and respiratory movements connected with breathing.
5. The chemical-sense irritation is different from that of smell, occurring only with irritating substances; it is felt on the mucous membrane.

III. In the imagination of smell the following points stand out:

1. Imagination per se was difficult for most subjects.
2. There were no changes in the imagined image such as were noted in optic or tactile imagination.
3. There was a definite admixture of optic imaginations.
4. The unpleasant odors could be imagined as more vivid and could be imagined more easily than the pleasant imaginations.
5. Imaginations were localized by the subject chiefly and with more reality in the nose, but were also localized in the head and in space.

IV. Experiments with after-effects and imagination of taste showed that:

1. The imagination of taste could easily be accomplished.
2. Visual images were noticeably present and directly connected with the object tasted.
3. The motor elements—chewing, swallowing and salivating—were intimately connected with the imagination of taste.

V. Forty cases of psychoses with olfactory hallucinations are cited and studied. The largest proportion of the conditions present were: schizophrenia; next, alcoholic hallucinoses; then, cerebral arteriosclerosis, epilepsy and sexual psychopathy. The outstanding characteristic of all hallucinated smells was their disagreeableness; they all contained organic elements and had direct or indirect references to the body and to bodily excrement. The group with organic disorders, as epilepsy and temporosphenoidal tumors, etc., with "uncinate fit" or isolated illusions or hallucinations of smell observed the same disagreeable qualities of smell as were noted in the group with functional psychoses.

VI. The significance of the decayed (organic) character in hallucinated smells is seen also in psychoanalyzed neurotic persons and in the analytic literature which deals with libido derivatives, instinctive drives, etc. Sensitivity to bad odors is probably a primitive defense reaction against dead bodies, against decay and disease in the body and against danger. Feelings about odors in the civilized state represent

also a repression of anal satisfaction, and in some way anality, interest and satisfaction with odors and homosexuality are equated. All these tendencies are shown in the psychotic patients. The relation of anal satisfaction to the defense mechanisms concerning decay could not be determined clearly.

VII. The obvious function of smell is to give a means of orientation concerning general spatial relationships, especially those related to food. In the deeper layers of consciousness it leads to heterosexual and anal satisfaction and gives orientation concerning the decay occurring in one's own body and the body of others. The reaction to these primitive tendencies is important for the formation of general social and esthetic attitudes.

VIII. The objectivity of smell, which is strikingly seen in the images, after-effects and imagination of smell, has an important bearing on its function as a social indicator. Smell is a purely objective sense and serves to maintain a relation with the external world. Smell helps one to pick out of the environment what is desired close to one or to reject and remove what one does not want near.

SPINAL CONCUSSION

A HISTOLOGIC STUDY OF TWO CASES

ROBERT S. BALDWIN, M.D.
CHICAGO

Spinal concussion has received various definitions during the last three quarters of a century. Hassin,[1] who reviewed the literature in 1923, pointed out that the term is reserved by some authors for instances of injury to the back in which anatomic changes are produced in the spinal cord without the intermediation of direct pressure on the cord, and that it is not used by them for direct injury to the cord from hemorrhage or tearing or from a sudden bending of the spine, which compresses the cord but leaves the canal thereafter unchanged.

Histologic study of the condition began when Schmaus [2] produced spinal concussion in rabbits and advanced with the subsequent confirmation of his work by Kirchgässer,[3] Spiller [4] and Jakob.[5] These investigators demonstrated degeneration of the nerve fibers and foci of softening in spinal cords unaffected by compression or hemorrhage. More accurate observations became possible after the experimental work in secondary degeneration by Jakob [5] and the histopathologic observations on human material by Hassin.[6]

Investigation of the pathologic anatomy of spinal concussion in man has progressed slowly. Schmaus [7] reviewed the literature up to 1889

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1. Hassin, G. B.: Concussion of the Spinal Cord, Arch. Neurol. & Psychiat. **10**:194 (Aug.) 1923.

2. Schmaus, H.: Beiträge zur pathologischen Anatomie der Rückenmarkserschütterung, Virchows Arch. f. path. Anat. **122**:326, 1890.

3. Kirchgässer, G.: Experimentelle Untersuchungen über Rückenmarkserschütterung, Deutsche Ztschr. f. Nervenh. **11**:406, 1897; **13**:422, 1899.

4. Spiller, W. G.: A Critical Summary of Recent Literature on Concussion of the Spinal Cord with Some Original Observations, Am. J. M. Sc. **118**:190, 1899.

5. Jakob, A.: Ueber die feinere Histologie der sekundären Faserdegeneration in der weissen Substanz des Rückenmarks, Histol. u. histopath. Arb. u. d. Grosshirnrinde **5**:1, 1912; Experimentelle Untersuchungen über die traumatischen Schädigungen des Zentralnervensystems, ibid. **5**:182, 1912.

6. Hassin, G. B.: Histopathological Changes in a Case of Amyotrophic Lateral Sclerosis, M. Rec. **91**:228 (Feb. 10) 1917; Studies in the Pathogenesis of Multiple Sclerosis, Arch. Neurol. & Psychiat. **7**:589 (May) 1922.

7. Schmaus, H.: Ueber den gegenwärtigen Stand der Lehre von der Commotio spinalis, München. med. Wchnschr. **46**:75, 1899; Ergebn. der allg. Path. u. path. Anat. **4**:676, 1899.

and found no satisfactory case. Since then Spiller,[8] Holmes[9] and Hassin[1] have observed in cords free from direct injury changes similar to those produced experimentally. This type of change has also been seen by Schmaus,[2] Holmes,[9] Hassin[10] and Davison and Keschner[11] in cords damaged directly by projectiles or fractures, but as these changes were observed several segments removed from the site of direct injury they were undoubtedly manifestations of concussion.

During 1933 there were four patients in the Cook County Hospital in whose cases a diagnosis of spinal concussion was made. I am reporting here two of the cases in which the spinal cords were studied histologically.

REPORT OF CASES

CASE 1.—J. P., a white man, aged 49, was admitted to the Cook County Hospital while semiconscious; a diagnosis of fracture of the spine and compression of the cord had been made. He complained of inability to move the lower extremities, inability to void and weakness and numbness of the upper extremities. The injuries resulted from an automobile accident seven hours previously, following which he had been unconscious for an hour or so.

Examination.—The patient was apparently unable to move the lower extremities; grasp was weak on both sides. The back was not deformed or tender. The bladder was distended to the umbilicus. The reflex activity was: knee and achilles jerks, exaggerated; biceps, triceps and radial jerks, absent; abdominal and cremasteric reflexes, absent; there was no response to plantar stimulation. Cutaneous sensibility was partially lost over the area supplied by the ulnar nerves, below the costal arch on the left side and below Poupart's ligament on the right side.

Diagnosis.—There were injury of the spinal cord at the level of the seventh to tenth thoracic segments and injury to the seventh and eighth cervical and first thoracic spinal nerve roots.

Laboratory Observations.—Extensive roentgen examination of the spinal column failed to reveal abnormalities of the bones. The spinal fluid was clear and colorless; the pressure was normal, and no block was demonstrated.

Course.—Within three days the deep reflexes of the lower extremities disappeared, and pressure sores appeared. The sensory changes varied from day to day. On the thirteenth day the patient noticed spontaneous movements of the lower extremities; however, the decubitus ulcers were more extensive, and urine from a retention catheter was cloudy. Four days later, the knee jerks were elicited, and the patient was capable of weak, voluntary movements of the thighs. At this time the sensory changes were stationary; they consisted of loss of pain and temperature sense below the costal arch on the left and below Poupart's ligament

8. Spiller, W. G.: Traumatic Lesions of the Spinal Cord Without Fracture of the Vertebrae, Univ. Pennsylvania M. Bull. **15**:483, 1903.

9. Holmes, G.: Spinal Injuries of Warfare, Brit. M. J. **2**:769, 1915.

10. Hassin, G. B.: Histopathologic Changes in Spinal Contusion, Arch. Neurol. & Psychiat. **13**:369 (March) 1925.

11. Davison, C., and Keschner, M.: Myelitic and Myelopathic Lesions: Traumatic Lesions of the Spinal Cord; a Clinicopathologic Study, Arch. Neurol. & Psychiat. **30**:326, 1933.

on the right, with preservation of tactile and pressure sense over these areas. The area of partial ulnar anesthesia persisted. The small muscles of the hands were markedly atrophied. During the next few days the patient lost voluntary control over the lower extremities, and the reflexes disappeared. Twenty-eight days after the accident he died of sepsis.

Necropsy.—The spinal column was intact; the vertebral canal was not narrowed; the cord was grossly normal; no signs of pressure were present over the dura, and only the blood vessels of the dura over the lower cervical and mid-thoracic regions were engorged.

Microscopic Observations: In sections made from the first and third thoracic segments, stained with toluidine blue, a minute softened area (fig. 1) was present in the posterior funiculus extending into the neck of the left posterior horn and

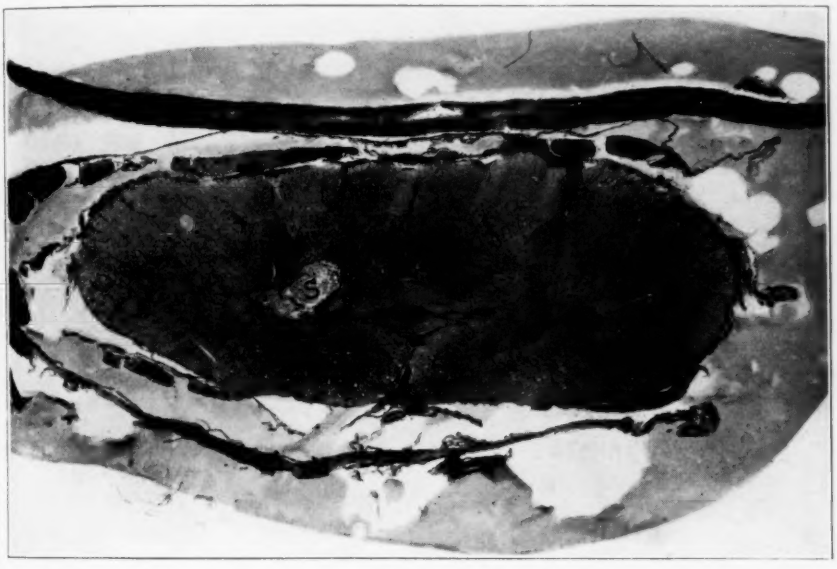

Fig. 1.—First thoracic segment. The posterior columns contain a focus of softening, *S*, which also invades the gray substance. This focus is pictured under higher magnification in figure 2. The anterolateral columns exhibit scattered vacuoles. Toluidine blue stain.

into the commissure. Numerous gitter cells (fig. 2) were scattered throughout the area of softening, which was surrounded by masses of lymphocytes and polymorphonuclear leukocytes. These cells are reactive, inflammatory cells, or manifestations of so-called sympathetic inflammation (Spielmeyer). No ganglion cells were found in the adjacent anterior horn (fig. 3).

In sections taken from other levels, stained according to the Alzheimer-Mann method, the majority of nerve fibers exhibited unchanged myelin sheaths and axons. A small number of vacuoles or empty spaces (fig. 1) were scattered throughout the white matter; they were more numerous in the anterolateral than in the posterior funiculus. They contained the gliogenous elements described by Jakob [5] as myeloclasts and myelophages. The former were small, granular bodies and contained pyknotic nuclei. The myelophages were larger and had eccentric

nuclei and a vacuolated cytoplasm containing fragments of myelin sheaths and axis-cylinders. In all of the sections there were astrocytes, mostly fibrillary. I was unable to make the distinction between myeloclasts and myelophages originating from oligodendroglia cells and those originating from microglia cells with the silver staining methods so successfully used by Cramer and Alpers [12] in animals.

In the cervical region, in addition to these manifestations of secondary degeneration, there was a marked glial reaction in the portion of the posterior funiculus corresponding to the area of softening previously described. The myelin in general exhibited degenerative changes which, as in polyneuritis, may be classified as primary. They were markedly swollen and tortuous and occasionally fragmented. Later they became, as in polyneuritis, transformed into lipoids, under-

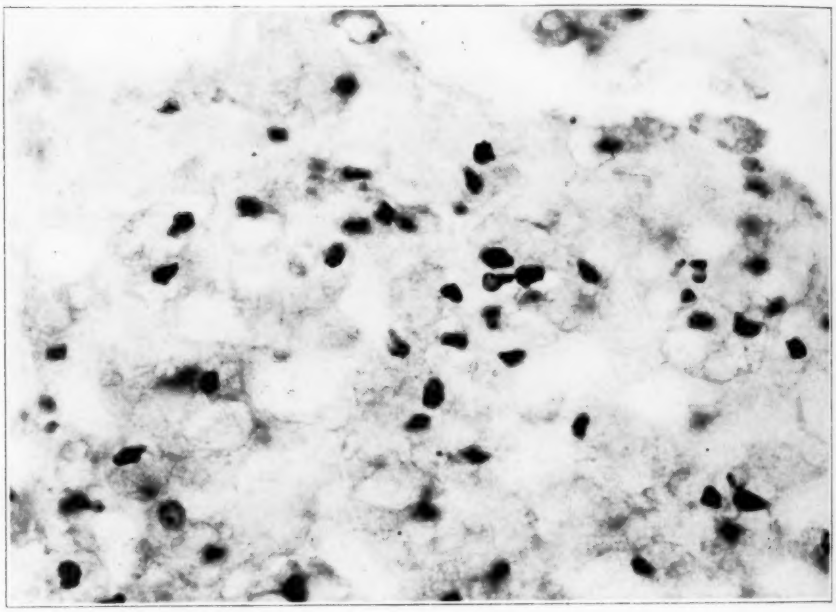

Fig. 2.—The focus *S* of figure 1 pictured under higher magnification. The numerous vacuolated cell bodies are gitter cells. Toluidine blue stain.

going secondary degeneration. Here it was thus possible to observe the changes of primary and secondary degeneration side by side.

Throughout the cord the walls of the blood vessels were intact. No blood pigment or red blood cells were observed outside the vessels except in the section from the tenth thoracic segment, in which small accumulations of apparently fresh red blood cells were scattered throughout the anterior horn on one side. But these accumulations of red blood cells were not associated with reactive glial changes and therefore must be considered as agonal. Schmaus,[13] who observed

12. Cramer, F., and Alpers, B. J.: The Functions of the Glia in Secondary Degeneration of the Spinal Cord, Arch. Path. **13**:23 (Jan.) 1932.

13. Schmaus, H.: Vorlesungen über die pathologische Anatomie des Rückenmarks, Wiesbaden, J. F. Bergmann, 1901, p. 388.

similar extravasations of blood in a patient who died twelve days after an injury, also considered them agonal.

Summary of the Microscopic Observations.—There were an area of softening in the posterior funiculus in the region of the first to third thoracic segments and vacuolated areas in the anterolateral columns at various levels, with early signs of secondary degeneration and the phenomena of primary nerve degeneration (swelling of myelin and of axons in some instances), and small agonal hemorrhages, which were rare.

Case 2.—C. G., a colored man, aged 56, was admitted to the Cook County Hospital with the history of a gunshot wound in the back three hours previously.

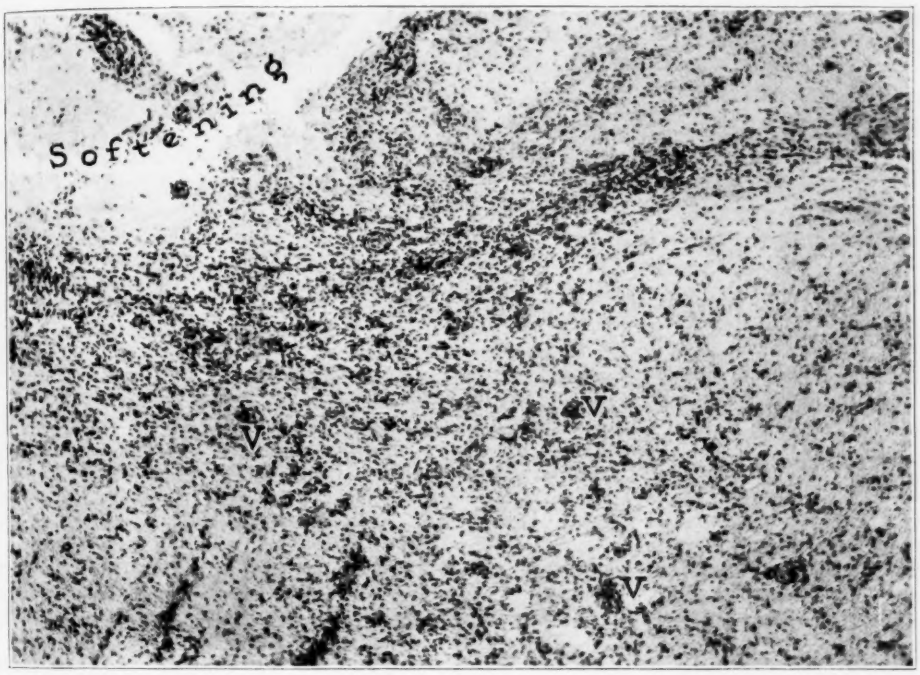

Fig. 3.—The area of the anterior horn of the spinal cord adjacent to the focus of softening. The blood vessels (*V*) are intact. The ganglion cells are absent. Toluidine blue stain.

He stated that the shot felled him to the ground immediately and rendered his lower extremities powerless. Complete flaccid paralysis of both lower extremities, paralysis of the bladder and rectum and cutaneous anesthesia extending up to Poupart's ligaments on both sides were noted. The wound of the back was over the spine of the eleventh thoracic vertebra, and the bullet could be palpated in the subcutaneous tissues of the right side of the chest at the level of the seventh rib in the midaxillary line. The abdomen was tense, tender and greatly distended.

Diagnosis.—A diagnosis of bullet wound of the back through the eleventh thoracic vertebra with section of the cord and possible puncture of the liver and transverse colon was made.

Fig. 4.—The second lumbar segment, showing marked vacuolation of the posterior and the marginal area of the right lateral columns; the left shows only a few vacuoles. Alzheimer-Mann stain.

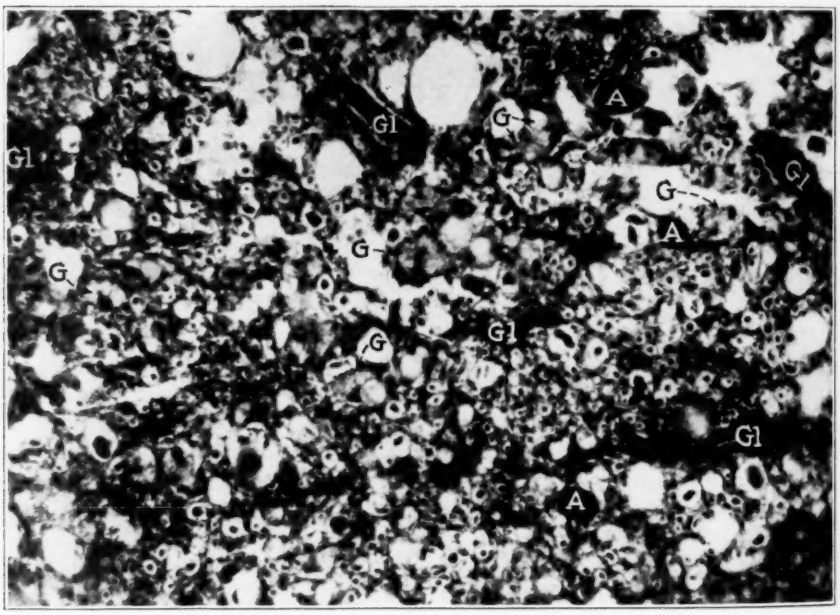

Fig. 5.—An area from the posterior columns of the lumbar region. Numerous degenerated nerve fibers are represented mainly by myelophages, which in the photomicrograph also resemble gitter cells (*G*). The large, empty spaces are vacuoles from which the contents (myelophages) dropped out. The large, dark, oblong bodies (*Gl*) are cytoplasmic astrocytes. The dark, round formations (*A*) are amyloid bodies. Alzheimer-Mann stain.

Laboratory Observations.—The contents of the stomach were free from macroscopic blood. The first specimen of urine was not abnormal, but on the ninth day cloudiness and shreds were seen; on the fourteenth day pus and blood were present.

Course.—The abdominal distention persisted, and the neurologic signs remained unchanged. The bladder was allowed to fill up and spill over. Fourteen days after the injury the patient died with a terminal picture of uremia: singultus, an odor of urea in the breath, uremic frost [14] and coma.

Necropsy.—The body of the eleventh thoracic vertebra was comminuted, but the vertebral canal was not encroached on, and the cord was grossly unchanged. Its configuration was normal, and the gray matter was sharply defined from the white substance. No hemorrhages, foci of softening or other anomalies were noted in the substance of the spinal cord or of its membranes.

Microscopic Observations: Sections stained by the Alzheimer-Mann method revealed degeneration of many nerve fibers singly and in groups in the various white fasciculi of the spinal cord, especially the posterior fasciculus (fig. 4). These areas were found at all levels and were especially numerous in the region of the first lumbar segment.

The degeneration of single nerve fibers appeared as cavities filled with myeloclasts and as large vacuolated cell bodies (myelophages) harboring fragments of myelin (fig. 5). In a section through the region of most marked alteration (first lumbar) there was a focus of softening, one fourth the size of the field of an oil immersion lens. Here there were numerous myeloclasts and myelophages, partially broken-down myelin sheaths and axis-cylinders. The substance of the cord bordering this area showed a marked glial reaction in the form of numerous fibrillary and cytoplasmic astrocytes (fig. 5), oligodendroglia and microglia cells. The walls of the blood vessels were intact in all sections, and no extravasations of blood were seen.

COMMENT

In the two cases recorded here, injuries of the back caused complete paraplegia, retention of urine and feces and marked sensory disturbances to total anesthesia, with no evidence of direct pressure on the spinal cord or hemorrhage into its substance. Similar to the case reported by Spiller,[8] case 1 presented a syringomyelic dissociation of sensibility, and the patient began to improve neurologically after thirteen days. It might be added here that, judging from the microscopic appearance of the cord, he would have recovered a considerable amount of function if the cutaneous lesions and infection of the bladder had not proved fatal. The course of the illness in case 2 was from the start that of a complete and rapidly fatal transverse lesion of the spinal cord.

The changes described here as primary and secondary degeneration of the spinal nerve fibers, foci of softening in the parenchyma of the cord and destruction of the ganglion cells can be considered as manifestations of concussion of the spinal cord. The breaking up of the nerve fibers, their secondary degeneration and the formation of cir-

14. Uremic frost is composed of dustlike deposits of urea crystals precipitated from the perspiration of patients in the last stages of uremia.

cumscribed foci of softening observed in these cases are entirely similar to the alterations experimentally produced by Schmaus.[2] The location of the focus of softening in case 1 in the ventral portion of the dorsal columns is characteristic. In many of the cords examined by Holmes[9] this region was similarly involved.

I shall not discuss the theories as to the pathogenesis of spinal concussion. This subject has been completely reviewed by Hassin.[1] I shall, however, mention the matter of hematomyelia, because it is frequently confused with concussion. Many clinicians[15] believe that temporary paralysis results from hemorrhage into the cord, and that restitution of function appears with resorption of the extravasated blood. This may occur, but I must say, with others (Schmaus,[13] Kirchgässer,[3] Claude and Lhermitte[16] and Hassin[10]) that hematomyelia as a pathologic and clinical entity is entirely different from concussion.

CONCLUSIONS

1. Trauma can produce permanent structural changes in the spinal cord without damaging the surrounding parts severely enough to cause their impingement on the cord.

2. These changes appear as degeneration of the cord, with foci of softening and no hemorrhages.

3. The changes, though most marked at the site of trauma, are usually scattered and may appear as destruction of single nerve fibers only.

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15. Donnet and Roussy: Un cas de lésion en foyer de la moelle lombaire (hématomyélie par traumatisme rachidien), abstr., Rev. neurol. **2**:987, 1915. Ballet, G.: Syndrome de Brown-Séquard par suite de commotion par éclatement d'obus sans plaie extérieure (hématomyélie vraisemblable par éclatement d'obus), Rev. neurol. **2**:768, 1915. Blake, J. B.: Hemorrhage into the Spinal Cord, Internat. Clin. **2**:86, 1918. Guillain, G.: L'hematomyélie des plongeurs, Bull. Acad. de méd., Paris **105**:996 (June 23) 1931. Marie, P., and Chatelin, C.: Un cas d'hematomyélie par éclatement d'obus à distance, Rev. neurol. **2**:777, 1915. Thornburn, W.: Cases of Injury to the Cervical Region of the Spinal Cord, Brain **9**:510, 1887.

16. Claude, H., and Lhermitte, J.: Gunshot Concussion of the Spinal Cord, Lancet **1**:67, 1919.

CHRONIC MANGANESE POISONING

REPORT OF A CASE, WITH AUTOPSY

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STANLEY COBB, M.D.

AND

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The clinical course and symptomatology of the rare industrial disease due to inhalation and ingestion of dusts containing manganese are now well known. Edsall and Drinker,[1] and Edsall, Wilbur and Drinker [2] had an opportunity to examine a larger series of these patients than has been available to other investigators, and they summarized their observations as follows:

As we have seen chronic manganese poisoning the following findings make the syndrome. We have numbered them in the most common order of appearance. It is difficult to emphasize in any written description the clearness with which the symptoms come out and the ease with which the diagnosis can be made.

1. A history of work in manganese dust for at least three months.

2. Languor and sleepiness.

3. Stolid, mask-like facies.

4. Low monotonous voice. Economical speech.

5. Muscular twitching, varying in degree from a fine tremor of the hands to gross rhythmical movements of the arms, legs, trunk and head.

6. Cramps in the calves and a complaint of stiffness in the muscles of the legs, the cramps usually coming on at night and being worse after a day of exertion.

7. Slight increase in tendon reflexes.

8. Ankle and patellar clonus. Frequently by stretching any of the muscles of the body it is possible to elicit rhythmical contractions. Romberg sign is inconstant: there is no incoordination.

9. Retropulsion and propulsion.

10. A peculiar slapping gait. The patient keeps as broad a base as possible, endeavoring involuntarily to avoid propulsion. The shoes are worn evenly, and we have not been able to convince ourselves of the pronounced tendency to walk

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From the Warren Museum and the Department of Neuropathology, Harvard Medical School, and the Department of Physiology, Harvard School of Public Health.

1. Edsall, D. L., and Drinker, C. K.: The Clinical Aspects of Chronic Manganese Poisoning, Contributions to Medical and Biological Research, Dedicated to Sir William Osler **1**:447, 1919.

2. Edsall, D. L.; Wilbur, F. P., and Drinker, C. K.: The Occurrence, Course and Prevention of Chronic Manganese Poisoning, J. Indust. Hyg. **1**:183, 1919.

on the region of the metatarsophalangeal joints, a feature strongly emphasized by von Jaksch.

11. Occasionally, uncontrollable laughter; less frequently crying.

12. Uniformly absent are any disturbances of deep or superficial sensation, eye changes, rectal, genito-urinary or gastro-intestinal disturbances, reactions of degeneration, blood, urine, and spinal fluid alterations. It is significant that, unlike lead, manganese produces no life-shortening degenerations. Seriously poisoned men are long-lived cripples. The metal apparently makes a very definite attack upon some non-vital portion of the neuromuscular system, destroys it thoroughly, if time for action is permitted, and leaves the victim quite well in every other respect.

We have never seen either the salivation or edema described in foreign cases.

Since 1919, when this statement was made, a number of cases have been reported. They have been found in the United States,[3] in England,[4] in Germany,[5] in France[6] and in Japan.[7] With almost unvarying regularity the disease has been due to inhalation of manganese dioxide in large amounts. The history in all the cases, back to Couper[8] in 1837, indicates that manganese is not a highly poisonous element, that it does not accumulate in the body, and that decided reduction of dust in the air breathed at once checks the appearance of cases. At the same time, it is equally apparent that patients with a well developed syndrome of manganese poisoning make but little recovery when removed from poisonous surroundings. They suffer no serious general impairment, as is the case in lead poisoning, and live for years as healthy but more or less helpless invalids. This makes them a charge on industry or on the state, and in the United States they may readily become serious medicolegal problems. This has become increasingly the case during the past ten years, owing to the appearance of cases of epidemic encephalitis (lethargica) with a parkinsonian syndrome. One of us (C. K. D.) has seen two such patients; both were but slightly exposed to manganese

3. (*a*) Davis, G. G., and Huey, W. B.: Chronic Manganese Poisoning: Two Cases, J. Indust. Hyg. **3**:231, 1921. (*b*) Gaylé, R. F. Jr.: Manganese Poisoning and Its Effect on the Central Nervous System, J. A. M. A. **85**:2008 (Dec. 26) 1925.

4. Charles, J. R.: Three Cases of Manganese Poisoning, J. Neurol. & Psychopath. **3**:262, 1922; Manganese Toxæmia; with Special Reference to the Effects of Liver Feeding, Brain **50**:30, 1927.

5. Flintzer, H.: Ueber gewerbliche Manganvergiftung, Arch. f. Psychiat. **93**:84, 1931. Baader, E. W.: Manganismus eines Trimmers, Zentralbl. f. Gewerbehyg. **19**:1, 1932. Mosheim, D.: Manganvergiftung bei Arbeitern aus der Mangan verarbeitenden Industrie, Klin. Wchnschr. **11**:1989, 1932.

6. Salmon and Planque: Intoxication par les minerais de manganèse, Ann. d'hyg. **11**:196, 1933.

7. Ashizawa, R.: Ueber einen Sektionsfall von chronischer Manganvergiftung, Jap. J. M. Sc. Tr. VIII, Int. Med., Pediat. & Psychiat. **1**:173, 1927.

8. Couper: Sur les effects du peroxide de manganèse, J. de Chim. méd. de pharm. et de toxicol. **3**:223, 1837.

and both showed typical epidemic encephalitis. One of these patients has become the center of a serious lawsuit.

In 1919, Edsall and Drinker [1] discussed the similarity of severe chronic manganese poisoning to paralysis agitans and to Wilson's disease (progressive lenticular degeneration). Lewy and Tiefenbach [9] produced changes in the corpus striatum of rabbits by giving large amounts of manganese salts. Mella [10] poisoned rhesus monkeys with manganese chloride; symptoms somewhat resembling those of manganese poisoning developed, and at autopsy there were diffuse cerebral changes which were most marked in the striatum and pallidum. Grünstein and Popowa [11] fed manganese salts to rabbits and obtained "a diffuse degenerative process in the nerve cells which reached its maximal intensity in the small cells of the caudate nucleus and putamen." These findings made it increasingly certain that the lesions of manganese poisoning in man would be found in the same region.

Autopsies on patients dying after manganese poisoning have been reported by Casamajor [12] and Gaylé,[3b] but Ashizawa,[7] in 1927, was the first to make a detailed report on the histopathology. His case was that of a man in whom typical manganese poisoning developed while he was working as a manganese dioxide grinder and who died three years after the onset of symptoms due to manganese. The conclusions and summary of Ashizawa are:

1. An examination of the pathologic changes in a case of chronic manganese poisoning disclosed diffuse changes in the central nervous system but with a definite degree of local intensification.

2. In general the changes were greater in the gray than in the white matter and were most pronounced in the midbrain.

3. The lesions in the pallidum where there was a marked loss of ganglion cells were striking. In contrast the nerve fibers were affected only in spots. Marked alterations were found in the putamen and nucleus caudatus, and the degeneration of the large ganglion cells was more pronounced than that of the small. The thin nerve fibers were lessened significantly in the striatum, but the thick fibers remained normal. Definite degeneration of Forel's bundle H_2 was noted.

4. The essential pathologic changes found in this case were in general the same as those described by Lewy and Tiefenbach [9] for rabbits and by Mella [10] for monkeys with experimental manganese poisoning. The only differences noted were that in both groups of animals the degenerative process in the parenchyma

9. Lewy, F. H., and Tiefenbach, L.: Die experimentelle Manganperoxyd-Encephalitis und ihre sekundäre Autoinfektion, Ztschr. f. d. ges. Neurol. u. Psychiat. **71**:303, 1921.

10. Mella, H.: The Experimental Production of Basal Ganglion Symptomatology in Macacus Rhesus, Arch. Neurol. & Psychiat. **11**:405 (April) 1924.

11. Grünstein, A. M., and Popowa, N.: Experimentelle Manganvergiftung, Arch. f. Psychiat. **87**:742, 1929.

12. Casamajor, L.: An Unusual Form of Mineral Poisoning Affecting the Nervous System: Manganese? J. A. M. A. **60**:646 (March 1) 1913.

was accompanied by proliferative changes in the glia and that in the rabbits the vessels were somewhat involved. In our case these changes were slight.

5. The marked parenchymatous damage found in our case had the same localization as the disease process in paralysis agitans and Wilson's disease or pseudosclerosis. These conditions present many symptoms similar to chronic manganese poisoning.

6. We may be assured that our case of chronic manganese poisoning was an instance of striopallidal disease.

REPORT OF A CASE

To Ashizawa's case we are able to add another, that of a man who first noticed symptoms after working for four years in a mill where manganese-containing fractions were separated from other ores. During this period the mill was extremely dusty; the air carried from 10 to 20 mg. of dust per cubic foot, and this dust contained from 9 to 12 per cent of manganese as soluble oxides and silicates.

The patient was first examined by one of us (C. K. D.) in 1918. The history and physical findings were:

History.—D. V. S., aged 55, a white man, married, a native American, was admitted on June 22, 1918, complaining of: "Walking bad. Can't go up hill or down." His father had died of old age, and his mother of dropsy. Five brothers were living and well. One brother had died of epilepsy. With this exception there was no history of nervous or mental disease in the family. The patient had been a temperate, healthy man. Typhoid fever, in 1898, was the sole illness prior to the present condition.

The patient had been a farmer until five years before, when he was employed at the mill as a cleaner and shortly after as a machine tender. He continued at this for three and a half years, when he shifted to a new mill as a motor tender. At both mills he was subjected to a heavy degree of dust inhalation. In September 1917, after four years of work in the dust, he noticed that he was getting clumsy and stiff, and soon he began to fall both forward and backward. He never had cramps in the calves or muscular twitching of any part. He was often awakened from sleep by a cramplike pain in the heels, but the pain was not centered in any muscle. There had been no change in disposition, i. e., no tendency to laugh or cry, and no headache or dizziness. With the onset of the symptoms the patient noted an increasing tendency to fall asleep at work. He was always tired and sleepy and felt it hard work to get up and down stairs at the mill. (He commented on the fact that two other men, who were becoming affected at the same time, also showed a great tendency to sleep.) This symptom had disappeared since his work was discontinued in February 1918. Since then he had done light work in his garden, but fell forward frequently; he called attention to the knees of his trousers which were torn by these falls. He could not work for more than an hour on account of fatigue and thought that his condition had remained stationary since he quit work.

Appetite and digestion were good. The patient had no constipation and no history of genito-urinary abnormality. He was alert mentally and commented intelligently on his own condition and on that of others with the disease. He expressed a strong opinion that another workman, J. B., so far unexamined, had the disease, and later observation confirmed the correctness of this view.

Physical Examination.—The patient was heavy and stood with feet apart; he was quite steady. His face was stolid and expressionless. No abnormalities of any sort were discovered until the examination of the extremities was reached.

Arms: There was no tremor of the hands, wasting or muscular twitching. The biceps and triceps reflexes were present and equal, but exaggerated. The finger-to-finger and finger-to-nose tests showed no incoordination. There were no astereognosis and no disturbances of sensation.

Legs: There was no wasting, paralysis, or muscular twitching. The knee jerks were present and equal, but exaggerated. The achilles reflexes were normal. There was no Babinski or Romberg sign. The gait was characteristic: The patient said that he had to "slap his whole foot on the ground to keep his balance"

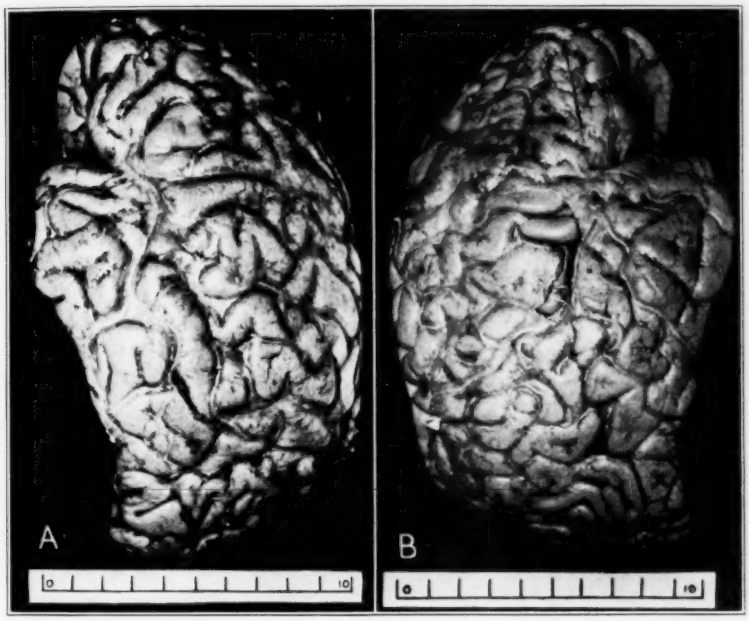

Fig. 1.—*A*, left lateral surface of the brain. Note the large vein over the mid-temporal lobe and the atrophy of the whole surface up to the occipital lobe; *B*, right lateral surface; compare with the left side. In this picture the atrophy is more patchy, involving only the vertex and frontal portions.

and he showed marked propulsion on attempting to walk on his toes; he fell on the second step backward. With the eyes shut he could walk forward and had never had trouble walking in the dark. There were no disturbances of sensation.

Laboratory Examinations: The results of laboratory examinations were normal throughout.

Course.—The patient was reexamined in 1921, 1922 and 1928. He never showed any improvement, and the condition did not advance. Pain in the heels and a degree of propulsion and fatigue which made it impossible to get about continued to be his chief complaints.

In January 1931, at the age of 69, he died of cardiorenal disease.

Necropsy.—Difficulty was experienced in securing permission for an autopsy, and the brain could not be removed until after the body had been embalmed. It was placed in formaldehyde U. S. P. 1:10 and sent to Boston for examination.[13] The abdominal and thoracic viscera were not examined.

Pathologic Report.—Without the dura, but retaining the leptomeninges, the brain weighed 1,440 Gm. The man's height was 6 feet (1.82 meters); thus by Tigges' formula the weight of the brain was normal.

The cerebral hemispheres were equal in length, 16.5 cm.; their width was 13 cm., and their thickness, 12 cm. The cerebellum seemed smaller than it should be in proportion to the brain, measuring 10.5 by 5.5 by from 2 to 3 cm. (fig. 1). There were asymmetrical convolutional patterns in the orbital portions of the

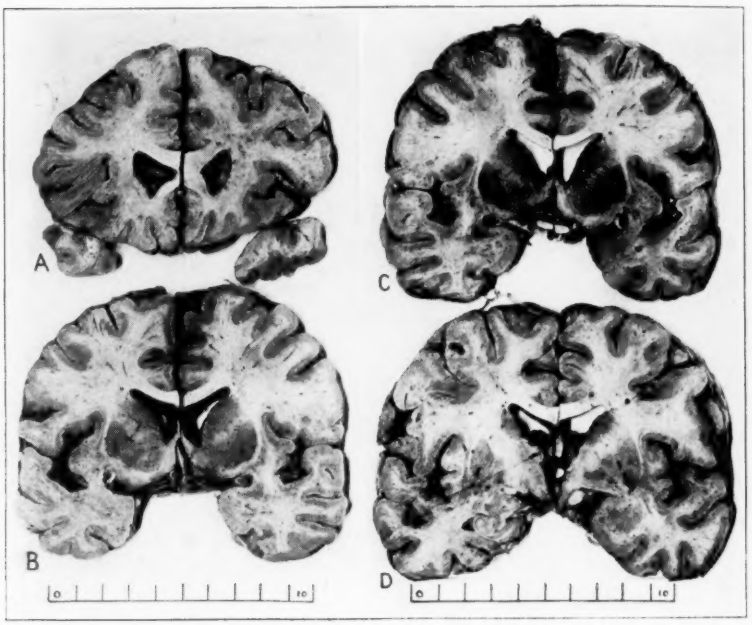

Fig. 2.—Slight dilatation and inequality of the ventricles are noted. The atrophy is most striking about the left third frontal convolution and island of Reil. In section *C* the atrophy is slight but general, especially in the upper temporal and lower frontal lobes. The ventricles are wide. In section *D* the basal ganglia show conspicuous atrophy, with resulting increase in the size of the ventricles, which are angular.

frontal lobes, in the temporal lobes, and in the occipital tips. The left hemisphere had a simpler type of convolution than the right in the second and third frontal gyri and also in the left parietal gyrus. The posterior third of each frontal lobe was simpler in type than the anterior third. The basal vessels showed minor anomalies and definite sclerosis (though not so advanced as is often seen at the age of 69). The cranial nerves were not remarkable.

13. Dr. Robert R. White performed the autopsy which provided the material presented in this study.

Frontal sections of the brain showed greater atrophy of the cortex on the left, and some indistinctness in the basal ganglia (fig. 2*C* and *D*). One slice of the brain, including both striate bodies and optic thalami (fig. 2*D*) was selected, prepared for sectioning *in toto* and stained by the Weigert myelin sheath method. For control a similar total section was procured from a woman, aged 55, who died of tuberculosis (fig. 4*A*). This was treated by similar methods. The section from the brain of the patient with manganese poisoning showed atrophy (fig. 4*B* and 5*A*). One could actually measure the decrease in size of the basal ganglia, which in different diameters were between 3 and 10 mm. smaller than in the control section (fig. 5*B*).[14]

Microscopic Examination: The tissues were fixed by embalming and were later preserved in formaldehyde. They were embedded in paraffin and stained with

Fig. 3.—In section *A* there is slight but general atrophy. The thalami are shrunken, and the ventricles are wide. In section *B* the white matter shows many congested vessels; the ventricles are dilated. In sections *C* and *D* there is marked hyperemia throughout the white matter. No atrophy is seen in these posterior sections.

cresyl violet, Mallory's connective tissue and Verhoeff's stain. In some cases other special stains were employed, e. g., Weigert's and phosphotungstic acid stain.

14. In reviewing the standard photographs of brains of the Massachusetts Department of Mental Diseases collection, made under the direction of the late Dr. E. E. Southard, twenty-six brains from patients varying in age from 70 to 92 years were used. Atrophy was present in some degree in all; the ventricles were seen to be dilated in eleven, though "dilated" is not to be used in an extreme sense and the condition would scarcely be recognized if one were not looking with a critical eye. In seven instances there was some change in the size of the basal ganglia, but in no case was there visible shrinking in all dimensions.

The choroid plexus showed cells on the outside of the tufts; they were mostly low and single but occasionally heaped up. Some tufts showed enlarged vessels, the walls measuring from 10 to 20 microns; some were thrombosed. The pia mater was definitely thickened. With an elastic stain it was striking to see the disarrangement of the elastic fibrils in many of the vessels: The inner elastic membrane was barely discernible, and fibrils could be found in clumps and snarls in the adventitia or farther out from the wall.

Sections from the cornu ammonis, stained with cresyl violet, Mallory's connective tissue and Verhoeff's stain showed a normal pia mater with dilated vessels.

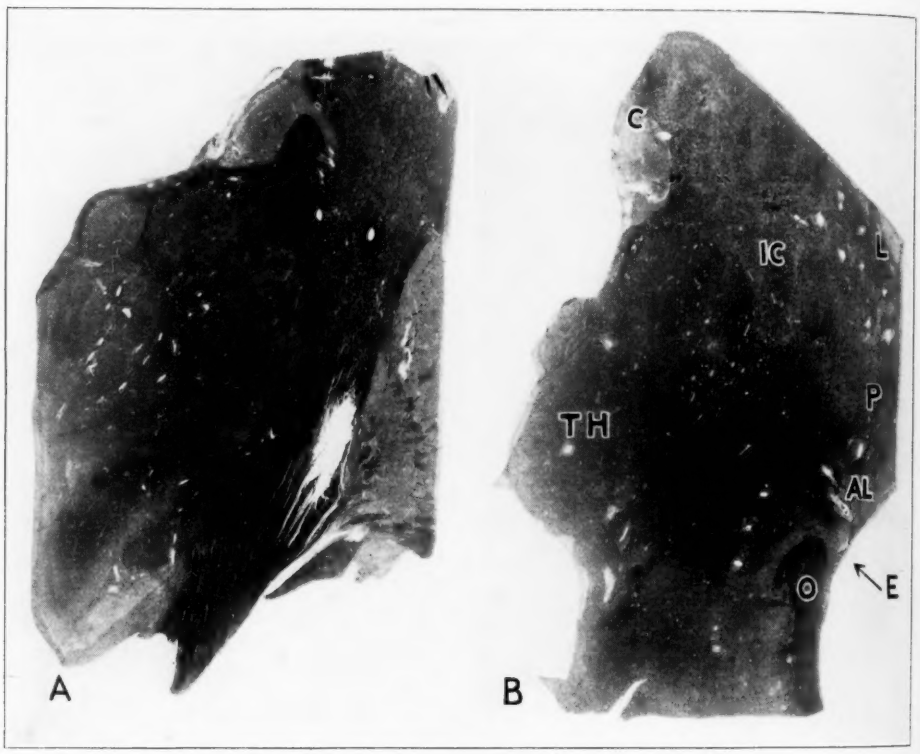

Fig. 4.—*A*, section through the basal ganglia of a normal brain for comparison with *B*. In *A* the boundaries of the different nuclei are well defined, and the internal capsule is clearly seen. *B*, basal ganglia of the brain of the patient with manganese poisoning. The internal capsule (*IC*) is wide in comparison with the shrunken thalamus (*TH*) and to the pallidum (*P*). The boundaries between the structures are blurred by gliosis. The ventricular surface of the thalamus shows many irregularities due to gliosis with contraction. *AL* represents the mesial of the ansa lenticularis; *O* is the optic tract. Between these at *E* is the site of the marked gliosis shown in figure 7.

The nerve cells stained rather deeply but retained their contour; the lower layers showed cells with satellites; otherwise this region was normal.

Sections from the left motor cortex showed fairly well preserved Betz cells. There was an increase of glia in the deeper laminae, and in the third layer there

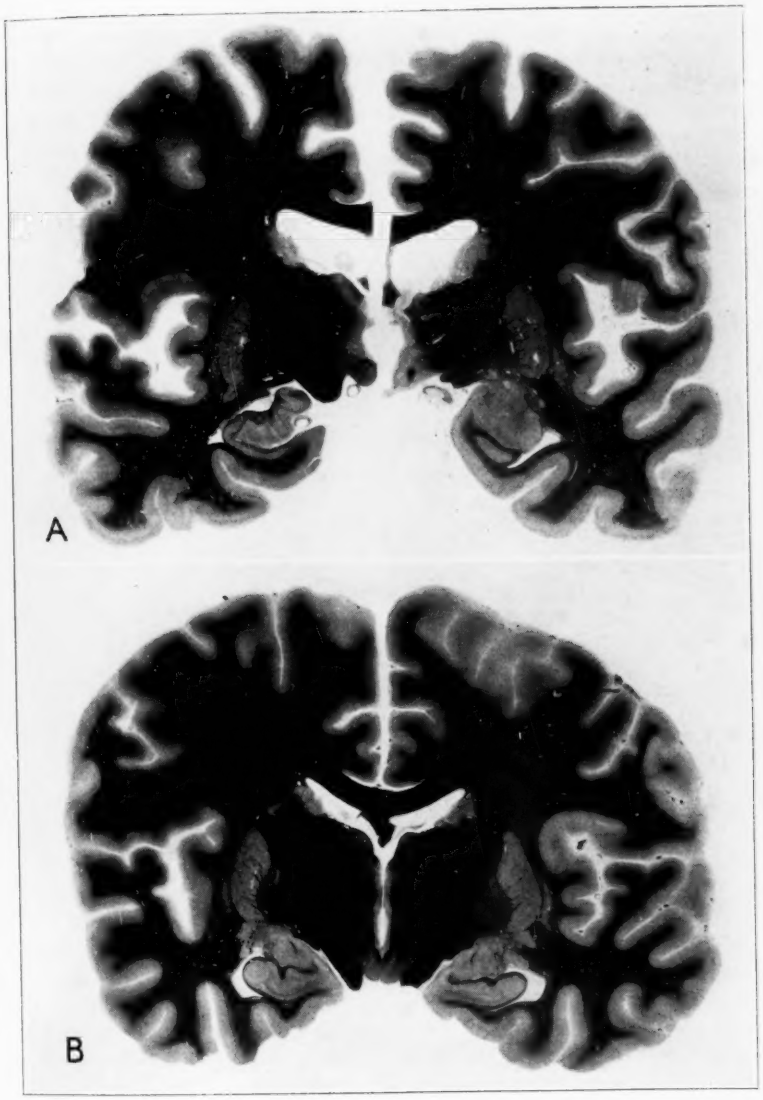

Fig. 5.—*A*, total section of brain of the patient with manganese poisoning, stained by the myelin sheath method. There is moderate cortical atrophy. The dilated ventricles result from the marked shrinkage of the basal ganglia. *B*, total section of a normal control (Weigert). Observe the small ventricles and the plump basal ganglia.

were some gaps in the nerve cells, filled with glia. In the white matter were some holes, probably due to swollen oligodendroglia. About some of the vessels were microglia laden with pigment. Similar sections from the control brain showed the swollen oligodendroglia and the gaps in lamina III.

Sections from the medulla oblongata were normal, except for slight subependymal gliosis and vacuolation of the ependyma.

Sections from the cerebellum showed Purkinje cells which were uneven in placement, swollen and paler than usual; the granule cells were well stained. The cells of the dentate nucleus stained in a range from a white lavender to purple; the pale ones were large, the dark ones shrunken. The vessels were distended but contained no blood (embalming?); there were occasional collections of perivascular cells.

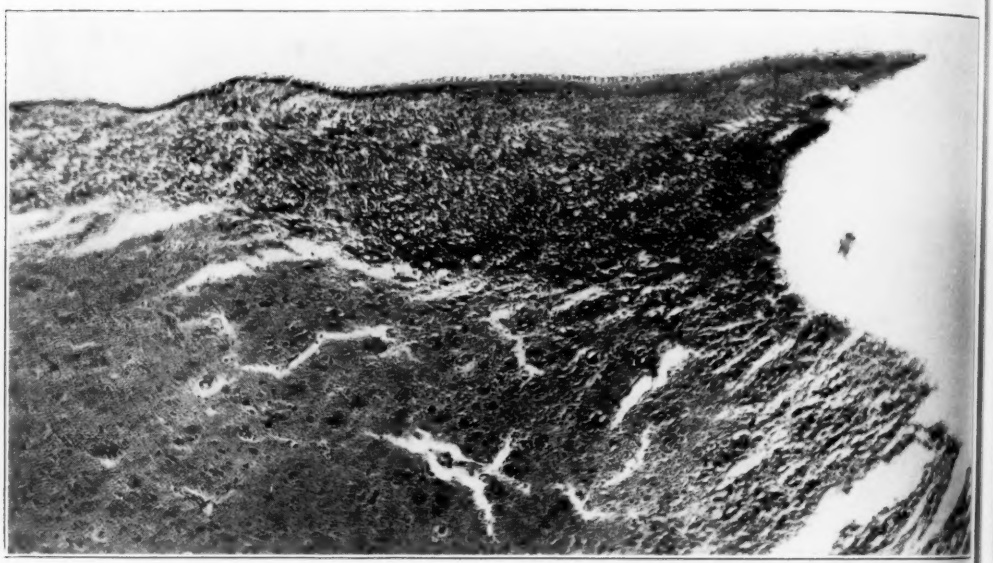

Fig. 6.—Higher magnification of part of the optic thalamus (× 100, phosphotungstic acid stain) shown in figure 4 *B*. There is a massive subependymal gliosis, and smaller islands are present beneath in the deeper parts of the nucleus.

The caudate nucleus was covered with a narrow ventricular ependyma, with vacuoles between the cells; beneath was an accumulation of glia cells packed in down to the level of the vascular bed. Some of the vascular walls were lined with black pigment. The cells of the caudate nucleus were remarkable—there was an excess of pigment in from one half to two thirds of them. They were surrounded by many glia cells, singly and in clusters of from three to seven. No nerve cells with normal Nissl bodies were seen. With phosphotungstic acid staining a similar section appeared densely blue, showing an excess of fibers.

In the optic thalamus the nerve cells were small and contained some pigment; everywhere glia cells were seen in excess, many as satellites. In the upper part of the structure there were many large nerve cells with pigment but fewer glia cells around them (fig. 6).

In the lenticular nucleus small nerve cells with satellites were seen in the upper part and there were many glia cells between the nerve cells. There was a marked

reduction in the number of nerve cells, so that whole fields of only glia occurred. In the lower part of the section pale but pigmented nerve cells were present, and around these was a marked increase of glia (fig. 7). Verhoeff's elastic stain showed walls of veins thickened, but without inflammatory cells; the elastic tissue in the arterioles was not well marked.

A comparison with the control specimens showed that most of these observations were abnormal. Especially important were the comparative cell counts which were undertaken in the basal ganglia to show their numbers in each portion. A number 2 ocular, tube length 160, and an apochromatic lens were used, and cells in ten

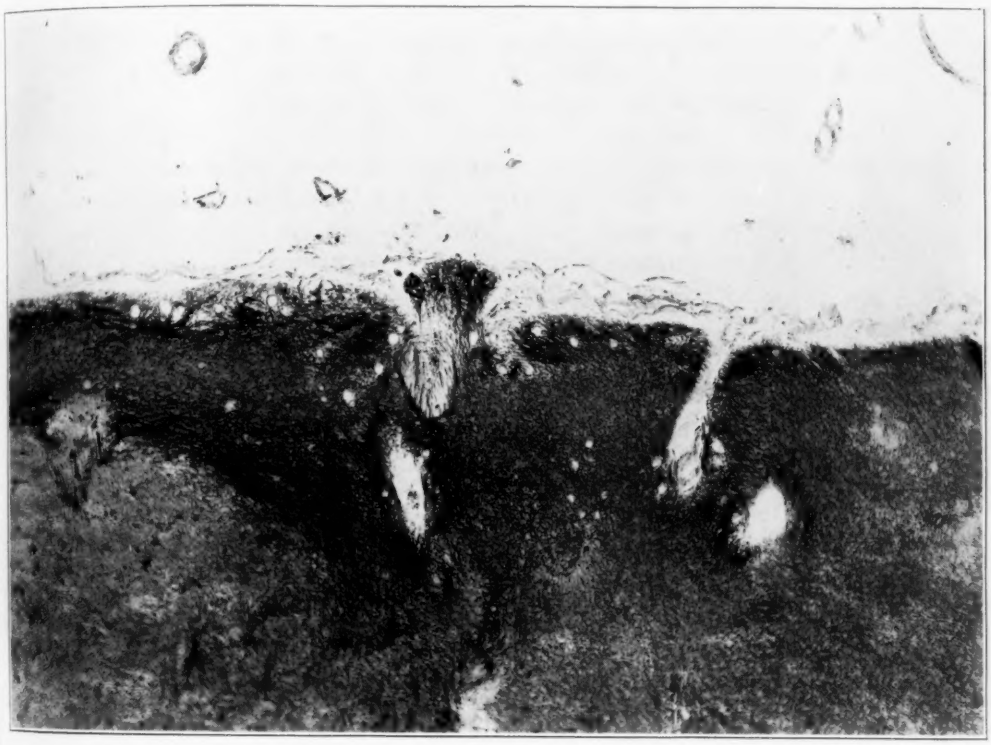

Fig. 7.—Detail in higher magnification (× 100) from figure 4*B*. Subependymal gliosis of the ventricular wall just below the area lenticularis (see fig. 4*B*). In the lower left hand corner of the section a part of the lenticular nucleus is seen which is also invaded by glia fibers.

fields were counted and averaged. In similar regions and in like manner ten fields in each locus were counted in the control brain. The results were as follows:

	Lenticular Nucleus	Caudate Nucleus	Optic Thalamus
Glia cells:			
Brain of patient with manganese poisoning...	127	129	110
Normal brain	69	72	54
Nerve cells:			
Brain of patient with manganese poisoning...	31	26	19
Normal brain	6	23	24

It will be noticed readily that there was a marked increase in the number of glia cells in the brain of the patient with manganese poisoning in all areas. In evaluating the difference in nerve cell counts it must be remembered that the nuclei were all shrunken, and the cells which remained could be concentrated by telescoping of tissue; this was best seen in the lenticular nucleus (fig. 8).

SUMMARY AND CONCLUSIONS

A man, aged 69, with manganese poisoning, evidenced by stiffness in the legs, gait, frequent falls, fatigability and sleepiness, died of cardiorenal disease fourteen years after the onset of the symptoms due to the occupational hazard. The brain showed atrophy over the vertex and lateral aspects but not at its base. This atrophy was somewhat more pronounced than that usually present at that age. On frontal section the atrophy was conspicuous, and there was dilatation of the lateral ventricles with marked shrinking of the basal ganglia.

Microscopic examination showed some diffuse cellular changes in the cerebral cortex and cerebellum, but the significant pathologic process was found in the basal ganglia, in which degeneration of nerve cells, satellitosis and gliosis were found. There was little difference of intensity in the process between the caudate nucleus, the putamen, the globus pallidus and the thalamus; in amount of gliosis, the optic thalamus led slightly, but marked focal scars were seen in the caudate nucleus and in the globus pallidus. The effect of the cellular damage noted in small sections was strikingly shown by the large total sections of the brain stained by the Weigert myelin sheath method. In those sections the shrinkage of the basal ganglia was measurable, distorting the anatomic outlines.

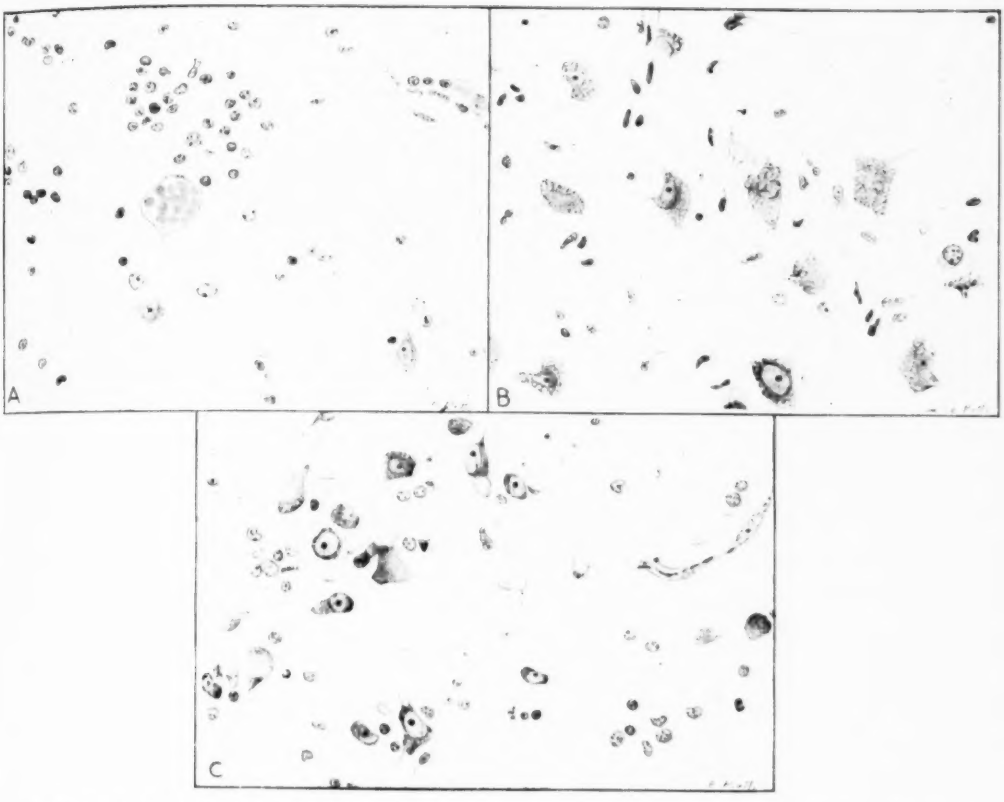

Fig. 8.—*A*, caudate nucleus stained with cresyl violet stain. This is a typical field from the devastated part of the nucleus; only one nerve cell is seen, and it is pale and swollen. There are groups of oligodendroglia cells and some astrocytes. There are no inflammatory cells surrounding the vessels. *B*, optic thalamus, cresyl violet stain. The nerve cells are distorted and their contours altered; some are faded, some polychromatic. In all but one the normal granules have disappeared, and the protoplasm has an almost vacuolated appearance. The vessels are proliferating. Note the preponderance of microglia, which was not apparent in the pictures of the caudate and lenticular nuclei. *C*, lenticular nucleus, cresyl violet stain. The field shows an irregular distribution of ganglion cells, many of which are in various stages of dissolution and not all of which are attended by satellites. Again the vessel wall has no inflammatory cells around it.

A NEW METHOD OF PRESERVING NORMAL AND PATHOLOGIC BRAIN TISSUE

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The gross specimens of normal and pathologic brain tissue employed for teaching and demonstration have certain disadvantages. They are usually preserved in solutions of formaldehyde or alcohol. A number of persons are so susceptible to the action of the former chemical that they cannot handle the material without gloves, while the rapid drying of the specimens preserved in the latter makes their close examination by any large number of persons at a time impracticable. In fact, the usual procedure is to view such specimens in the jars in which they are kept and to hand about the jars at meetings or in classrooms.

The method perfected in the Neurological Laboratories of Columbia University obviates these disadvantages. The preparations are dry, and they possess certain esthetic qualities, so that instead of arousing in the examiner a sense of aversion they do the opposite—they arouse his sense of curiosity. The slight translucency of their surfaces accentuates in a number of these preparations the differences in structure, so that one actually sees more in them than in the specimens preserved in formaldehyde or alcohol.

TECHNIC

The technic consists in the infiltration of the tissue with the insoluble, infusible and practically noninflammable synthetic resinoids of the phenol series.

Infiltration.—Slabs of brain tissue of any thickness, preserved in formaldehyde, are placed for about a week in a 40 per cent solution of formaldehyde. They are then put in a solution of synthetic resinoids of the following composition: phenol, 4,530 Gm.; trioxymethylene, 1,684 Gm.; sodium hydroxide, 15 Gm., and salicylic acid, 15 Gm. The phenol crystals are melted by heating, and to this liquid is added the sodium hydroxide and salicylic acid, previously dissolved in a few cubic centimeters of water, and next the trioxymethylene.

Great caution must be exercised in the preparation of the resinoid solution. Its temperature begins to rise after the addition of the trioxymethylene, at first gradually, then with increasing rapidity until, in the course of an hour or two, depending on the temperature of the room, unless precautions are taken it fulminates, evolving great quantities of formaldehyde gas. Once the solution begins to boil, the best that one can do is to pour into the container a quantity of cold water, empty the whole into the sink and leave the faucets open. If fulmination has proceeded to the stage at which the solution becomes viscous, the chances of stopping up the plumbing should be borne in mind.

A practical way of avoiding this is to place the jar in which the ingredients are mixed in a large bucket full of running cool water. The ingredients will

crystallize in the course of an hour or two, but the jar should be kept in the running water for twenty-four hours, after which the danger of fulmination is passed. The ingredients will dissolve and combine in the course of a week or two. The process may be expedited without danger after the first twenty-four hours by placing the container in a bucket of warm water.

When ready, the solution is clear, transparent and of the reddish-yellow color of light amber.

Slices of brain tissue, 15 mm. thick, become thoroughly infiltrated in this solution, at room temperature, in from ten days to two weeks; thicker pieces become infiltrated in a correspondingly longer time. The specimens are now soft and somewhat friable. When placed on a plate of glass and viewed over an electric light, slabs ½ inch (1.27 cm.) thick are seen to be translucent throughout.

Polymerization.—The specimens are then removed from the solution, blotted between paper towels and placed in a thermostat at an initial temperature of

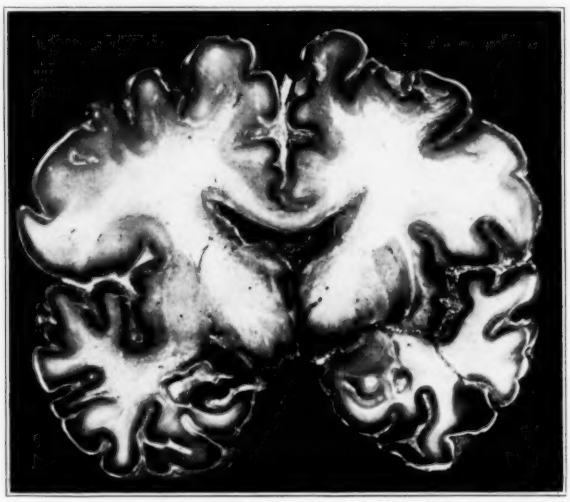

Fig. 1.—Normal brain, coronal section. Note the clear differentiation of the structures.

36 C., which is gradually raised so that at the end of a month it will have reached 80 or 85 C. The preparations are then dry and so hard that they cannot be indented with the finger-nail.

Finishing.—Thin slabs may then be mounted on fiber board in one of the many standard ways of mounting. Thicker slabs or irregular large pieces need no mounting.

The slabs are then ground down on a garnet or carborundum sanding-disk until the surfaces are flat. They are then polished on a buffing wheel, at first with powdered pumice and water, then with rouge. The resulting surfaces are bright and glossy.

Developing.—In a number of preparations the differentiation of the several structures is good at this stage. In most cases, however, the color is unsatisfactory, generally a dark purple, which is, in some instances, almost black, and the outlines of the various structures are diffuse and ill defined. In such cases the preparations must be further treated by a process of developing in a solution composed of 0.1 per cent sodium hydroxide and 2 per cent salicylic acid. The preparations are

Fig. 2.—Intracerebral tumor, showing clearcut differentiation.

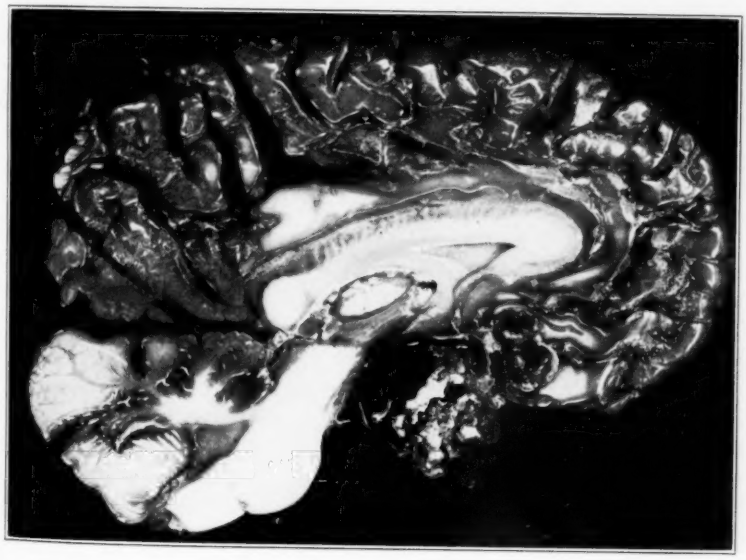

Fig. 3.—Extracerebral tumor.

boiled in this solution for a variable time, from fifteen minutes to two or three hours, depending on their condition, until the surfaces are covered with a yellow film. The latter is buffed off with powdered pumice and water until the structures which it covers appear clearly outlined.

This developing solution may be used a number of times and becomes increasingly effective with continued boiling. On cooling, the constituents crystallize. The discolored supernatant liquid may then be poured off and fresh water added to nearly the original volume.

COMMENT

The entire process, carried out on a small scale, appears to be rather difficult and complicated. Done on a large scale, as it is in the laboratories, it is simple. A large hotbox, in which the source of heat is an electric plate placed at the bottom, has different temperatures at different levels. By lowering the shelves on which the preparations are placed a given distance each day, the temperature is gradually increased until the process is completed.

A large number of different preparations can be treated in a single container by placing each in a gauze bag. This is true of the process of infiltration as well as that of developing.

In the grinding of the preparations it is rather important to avoid the resulting fine dust. This is accomplished by enclosing the entire sanding outfit in a tight box with a glass top and two openings in elastic membranes for the operator's hands.

As the solution of the resinoids is used over and over again it becomes diluted. It is not an easy matter to extract the water from two such powerful dehydrants as phenol and formaldehyde. Gelatin, which is so effective in extracting water from dilute alcohol, is useless in the solution in question, for it is hardened, ceases to absorb water and does not swell. Boiled and dried starch or rice grains are effective in removing water from the solution; both hold in their interstices a large quantity of the resinoid, however, which is wasted when these absorbents are removed. A practical way out of the difficulty, which I have followed successfully, is the use of beans for that purpose. A number of peeled dried lima beans, in a gauze bag, placed in the diluted resinoid will swell in the course of a day or two by the absorption of water. The bag of beans can then be washed in running water for twenty-four hours, dried and used again and again.

In preparing large numbers of slabs of brain tissue for the use of classes, it has been found practical to reinforce them in the following manner:

In the stage of polymerization, at which the specimens are of a rubbery consistency, stout brass wires are inserted in a number of places. Large open crevices, such as the great longitudinal and the horizontal fissures, are filled in with a paste made from the grindings mixed with the solution of the resinoids.

The three photographs are examples of the average preparations.

ATOPY AS A CAUSE OF EPILEPSY

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COLUMBUS, OHIO

With recent knowledge about the cerebrospinal fluid, water metabolism and the anomalous dural venous sinuses, an entirely new picture appears in the study of epilepsy. To this hopeful outlook the development of allergy is making its contribution. It is my purpose to discuss a small but definite group of cases, which heretofore have been indistinguishable from other cases of essential, or idiopathic, epilepsy, occurring in persons with an inherited tendency to specific, abnormal reactions to certain inhalants, contactants and, more especially, foodstuffs. It is to be understood that I use the terms in the field of allergy in a rather strict sense. Specific hypersensitiveness experimentally induced in animals is called "anaphylaxis." Specific hypersensitiveness occurring spontaneously in man is called "allergy." Allergy is divided into serum sickness, bacterial allergy, contact dermatitis and atopy. Atopy is that form of allergy which is controlled by inheritance, and is the form with which I am concerned here.

Since almost every other possible theory has been evoked to explain epilepsy, it is to be expected that the seizures would suggest a hypersensitiveness. This is less surprising when one considers that the underlying pathologic process of an allergic reaction, i. e., spasm of smooth muscles, increased permeability of capillaries and edema of tissues, could well explain how these seizures are produced.

In 1923, the late Luther P. Howell reported fourteen cases of epileptic patients who either had given positive cutaneous reactions to certain food proteins or were seized with fits on eating certain foods. I saw three of these patients nine years afterward. They had remained free from attacks except when they encountered the specific foodstuffs to which they were sensitive.

Enough evidence has been brought forward in the literature to show that atopic phenomena are present in a small percentage of cases. An attempt to invade this field of study by the allergist must be carefully controlled, because it is apparent that atopy can be only one of many causes of epilepsy. Still, if careful study reveals no significant abnormalities the condition may be called essential or idiopathic, and if in this more limited group one can find now and then a case explainable on an atopic basis it is well worth the effort.

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The first step of the allergist is to classify the case as (1) atopic or (2) nonatopic. There are four essential criteria for the recognition of atopy: (1) a familial and (2) a personal history of asthma, hay fever, vasomotor rhinitis, hives, eczema, migraine-like headaches and other allied conditions; (3) eosinophilia in the blood preceding and during the attacks, and (4) positive reactions to cutaneous tests for sensitiveness to protein. The positive reactions are not always of clinical significance in the consideration of the seizures, but may be the record of earlier difficulties with food or breathing.

If (1) a family history of atopic manifestations or (2) a personal history revealing other atopic reactions in the patient himself is not obtained, the patient is returned to the neurologist. But if such a history is obtained, the remaining criteria of (3) eosinophilia and (4) positive cutaneous reactions are sought. With these rigid requirements one may eliminate the instances (at least 25 per cent) of atopic persons whose family histories are negative for allergic manifestations and of patients who do not give positive cutaneous reactions. Later, when the thesis of this paper has been established, the allergist may assume the less rigid requirements demanded for pollen hay fever.

There are two methods of service in identifying the offending substances in atopy: skin tests and clinical trial. These are supplementary to each other.

SKIN TESTS

Skin tests are applied in (1) the direct and (2) the indirect manner.

1. *Direct Method.*—This is used in three ways: (*a*) the patch test (not of use in these cases); (*b*) the scratch test, and (*c*) the intradermal tests (the protein extract in a safe solution is injected into the skin).

The difficulties encountered in the interpretation of the reactions to direct skin tests have been classified as follows: (1) false negative reactions; i. e., the patient gives negative reactions to substances to which he is clinically sensitive. It is therefore of fundamental importance that the materials used for the testing be known to be potent, and that the protein used be chemically as nearly as possible like that with which the sensitized tissue of the patient usually comes in contact; (2) false positive reactions; i. e., the patient gives a negative reaction clinically to a substance, the extract of which produces a reaction which I have called positive. Extracts of certain foods are troublesome in this regard. A positive reaction may represent a sensitization other than that which the physician is seeking. It may represent childhood eczema, asthma or some other past condition with which the patient is now in equilibrium or it may represent a future condition.

2. *Indirect Method.*—This method of skin testing was devised by Walzer. It attempts to detect the presence of the specific antibodies of atopy (reagins) in the circulating blood of the patient by means of tests performed, not on the patient himself, but on certain sites on the skin of a normal subject into whom some of the patient's serum has previously been injected. In dealing with cases of seizures which occur only at rather long intervals, this method of testing is of inestimable value. This is because the use of the elimination-substitution-

identification diets for the purpose of detecting the offenders has, in dealing with epilepsy, two distinct drawbacks. In the first place, since the atopic person does not invariably exhibit symptoms when exposed to the substance to which he is sensitive, it usually takes months and even years to prove or disprove the offensiveness of the proteins which caused the positive reactions. Under these circumstances it becomes impossible to control the average patient. Only the most intelligent will continue their cooperation long enough. In the second place, one will never detect and establish the effect of the proteins which are absorbed by inhalation or contact.

TREATMENT

The treatment may be outlined as follows:

Specific Treatment.—1. This includes attempts to remove the specific excitant from the patient's food or environment. This is the method of choice with foods, especially in children and young adults when there is a tendency to gain tolerance by a period of avoidance ("outgrowing it"). If the substances for which there are circulating antibodies (reagins) have been identified by indirect testing, this method of treatment may be carried out at once. The diagnostic use of the elimination-substitution dietary regimen serves not only to identify the offenders but to keep the patient free from seizures.

2. This method attempts to lessen the degree of sensitivity or to raise the tolerance (sometimes erroneously called desensitization) by graduated injections of a standardized preparation of the specific substances to which the patient is sensitive. In general, however, it may be said that foods are the common cause, and attempts to raise the tolerance by the injection method are never as successful with foods as with inhalants. Thus the dependence in most instances must be on a program of elimination. When the excitant is not a food, the patient must be kept from coming in contact with it. Instances are on record in which the elimination of offending tobacco resulted in relief.

3. This method attempts to lessen the degree of sensitivity by feeding the offending food in minute, measured doses, which are gradually increased in amount until a complete tolerance is gained. This is only worthy of trial as a sort of "last straw," as it has not proved successful in many instances, whether dealing with the whole food, the protein extract of the food or its polypeptides after partial digestion.

Nonspecific Treatment.—This method is concerned with constitutional measures including attempts to lessen the degree of sensitivity of the patient: (1) by stabilizing his "atopic equilibrium" through removing the secondary, or nonspecific, aggravating factors, e. g., changes in temperature and exposure, emotional disturbances, constipation, fatigue and exhaustion, malnutrition, focal and acute infection and disorders of the ductless glands, and (2) by changing his physiochemical balance to a different level, by administering drugs, to overcome alkalosis, to induce hypo-allergiosis, or to decrease the irritability of the cerebral cortex, or by giving vitamin A.

REPORT OF CASES

CASE 1.—A boy, aged 10 years, was brought to me three years before the writing of this report, because of seizures of the grand mal type. A complete examination failed to reveal any organic disorder. The family history revealed a maternal grandfather with hay fever, a maternal grandmother with frequent and persistent urticaria and attacks of migraine and a mother who had eczema as a child and

at the time of examination had mucous colitis, persistent canker sores and migraine. A great-grandfather had attacks of epilepsy which did not begin until he was past middle life and which were always thought to be the result of trauma. The patient himself presented no other manifestations of atopy.

On the day following an attack, blood smears revealed eosinophilia of 5 per cent. The application of 109 skin tests by the scratch method gave the following reactions: sweet potato ++, fish +++ and milk ±.

On a strict elimination diet, the patient became free from seizures. The exhibition of offenders, however, resulted in their recurrence.

CASE 2.—A man, aged 30, consulted me three years and three months before the writing of this report, because of a typical seizure of the grand mal type. In the family of both parents were instances of hay fever, asthma, migraine, hives, persistent canker sores of the mouth, epilepsy (petit mal in the mother), eczema and mucous colitis. The personal history was unimportant except for the present complaint. Four hours after a seizure there was an eosinophilia of 6 per cent in the differential blood smear. There were positive reactions to intradermal skin tests for wheat, rice, milk, eggs, peas, mustard and spinach. Rice, peas, mustard and spinach were not exhibited in the course of treatment, but the exhibition of wheat, milk and eggs on several occasions was followed by a seizure. No seizures occurred at any other time than on the inclusion of these foods in the diet of the patient. By paying strict attention to diet he can go about his daily work in good spirits.

CASE 3.—A man, aged 36, consulted me on March 7, 1932, because of seizures of the grand mal type. The attacks started three months before, and were severe and frequent. The family had been given, confidentially, a hopeless prognosis. Complete study failed to reveal any physical abnormalities. Inquiry into the family history revealed that the mother had migraine. The patient's own history revealed mild vasomotor rhinitis. Blood smears shortly after a seizure showed eosinophilia.

The results of intradermal skin tests were: positive reaction to goat hair, house dust and orris root (vasomotor rhinitis?); tomatoes, +; corn, +; wheat, ±; red pepper, +; Brazil-nut, +, and cherry, ±.

The patient was placed on a strict regimen of elimination. As he broke his leg it was possible to control the diet absolutely. It was clear that cabbage, tomatoes, and beans, when placed in the diet, would be followed by seizures. During the following two years this patient, except for the time when he was recovering from the fracture, worked hard every day. There were only two series of attacks which were not easily explained on the basis of diet, but which might have been induced by extreme fatigue.

CASE 4.—A boy, aged 15, for several months had many attacks of grand mal, and fell off the roof of a barn and injured himself in one attack. The family history exhibited the following atopic manifestations: The father had asthma and ragweed hay fever, many attacks of hives and migraine, and had shown idiosyncrasies to drugs to the age of 30. A paternal aunt had grand mal seizures. The maternal grandmother had migraine.

Positive reactions to intradermal skin tests were caused by: goose feathers, +++; dog dander, ++ (somewhat delayed); carrot, +; rye, +; spinach, ++; white potato, +++; lettuce, +; asparagus, +, and coffee, ++.

The patient left immediately after examination for a private school in the South. No attempt was made to carry out suggestions, and no improvement was noted.

CASE 5.—During the last week in August 1932, a woman, aged 29, was sent to me because of a convulsive seizure during the height of her fever. She and her husband drove 350 miles into Canada, where she was free from symptoms. They stayed two days in this zone but continued to drive. They then started home; when they entered Ohio the hay fever returned in full force. When she reached home she was nearly exhausted. That night the husband was awakened to find her unconscious and in a convulsive seizure; she had bitten her tongue.

A complete study failed to reveal any physical abnormalities. The family history revealed atopic manifestations. The maternal grandmother and her sister were victims of asthma. The grandmother also had mucous colitis. The mother was a victim of migraine. A brother had ragweed hay fever and horse asthma. The patient's personal history also revealed atopy, since in addition to the immediate condition she had both grass and weed hay fever and migraine. Blood smears on the third day following an attack showed 6 per cent eosinophilia. Reactions to skin tests by the scratch method were as follows: brewer's yeast, ++; celery, +; haddock, +; lemon, +; beef, ++; rye, ++; wheat, +++; mackerel, +; green pepper, +; salmon, +++; cantaloup, ++; tomato, ++; chocolate, +++; house dust, +++; timothy grass pollen, +; ragweed pollen, ++. An elimination diet was prescribed and the spasms did not return, even when she forsook the diet a few weeks later.

CASE 6.—A man, aged 30, consulted me sixteen months before the writing of this report, because of seizures of the petit mal type. Both the family and the personal history revealed atopy. Blood smears showed an eosinophilia of 5 per cent, and many skin tests with foods and tobacco gave positive results. The pollens, contactants and other inhalants gave negative results. The use of Rowe's no. 1 elimination diet brought prompt relief. The exhibition of chocolate, eggs and cabbage was invariably followed by attacks.

CASE 7.—A boy, aged 16, consulted me because of frequent attacks of grand mal for the preceding five weeks. Complete clinical, roentgen ray and laboratory studies failed to reveal any physical abnormality. The patient had been advised to take phenobarbital because of a diagnosis of essential epilepsy. Inquiry into the family history revealed that the mother had fainting (?) spells as a girl, and had migraine and vasomotor rhinitis. A sister had had hives on several occasions. The patient's personal history revealed a nettle-rash on eating corn, in addition to the present condition.

Intradermal skin tests with the usual extracts gave the following results: tomato, +; cornmeal, ++; cabbage, +; lemon, +; mackerel, ++; salmon, +; cantaloup, +; rice, +++; wheat, ++; house dust, +++; dog dander, +; goat dander, +, and goose feathers, ++.

On an elimination program the patient immediately stopped having seizures until the Christmas holidays, when indulgence in foods brought a recurrence. The elimination diet brought relief until Easter, when indulgence again caused a recurrence. Since then no trouble has occurred.

CASE 8.—A married woman, aged 30, whose family history was not obtainable, had grand mal seizures for five years. She presented no other atopic manifestation. There was an eosinophilia in the blood of 6 per cent. Intradermal skin tests for chocolate, carrot, potato, tomato, oats, wheat, lemon, cantaloup, haddock, mackerel, salmon and asparagus gave positive results. In the presence of bad mental hygiene, excessive smoking and a refusal to live on a restricted diet, nothing could be done with the patient. It would have been better if we

had not made the tests. This case illustrates the folly of an allergist accepting all patients who are sent to him for skin tests.

CASE 9.—A girl, aged 15, consulted me because, from time to time during the previous two years, she had been having typical grand mal attacks. Inquiry into the family history revealed that the maternal grandmother had vasomotor rhinitis, a maternal cousin was a victim of hives and another maternal cousin had been troubled with canker cores. There was no history of epilepsy or migraine in the family. The personal history suggested a previous vasomotor rhinitis, which was not particularly active at the time of consultation.

Intradermal skin tests with eighty of the more common offending proteins revealed strong reactions to dog dander and feathers (cause of the coryza?) and to wheat, oats and spinach. Avoidance of the inhalants indicated and the use of an elimination diet brought relief. She did not try to use the substances to which she gave a positive reaction. She added red cherries to her diet and on the second day had an attack. She had not been tested for these, but they were eliminated.

CASE 10.—A boy, aged 12, who was first seen on Nov. 15, 1933, had had typical grand mal attacks for the preceding six years. For several months he had been seized from four to nine times in every twenty-four hours. In August, he was studied at a good neurologic clinic, but nothing definite was found.

Phenobarbital was administered, with no improvement. The family history revealed atopic manifestations. The patient also had vasomotor rhinitis and a paternal first cousin was epileptic.

Intradermal skin tests revealed: house dust, ++; feathers, +++; sheep wool, ++; kidney beans, +; wheat (marked delay); apple, +; beets, +; peppermint, +; egg, +; peas, +; oats, +; mackerel, +; haddock, +, and milk, +. An elimination diet resulted in cessation of the attacks within ten days. An attack occurred during Christmas week, when the foods causing positive skin reactions were returned to the diet. Milk, eggs, cherries and spinach were associated with recurrences; otherwise the boy was free from epilepsy and gained weight.

SUMMARY

Clinical evidence is brought forward to show that, by the application of the criteria necessary for the recognition of atopy, cases of essential epilepsy can be selected in which atopy is of fundamental importance, and that if these selected patients are not allowed to come in contact with the specifically offending materials seizures do not occur.

CEREBROSPINAL HYDRODYNAMICS

IV. CLINICAL EXPERIMENTAL STUDIES

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In the initial article of this series [1] evidence was presented to show that in fresh human cadavers the normal ventriculosubarachnoid spaces of the brain remain in free communication with the subarachnoid spaces of the spinal cord during various experimental changes in the pressure of the subarachnoid fluid, and that such variations of pressure, however rapid, do not produce any apparent movement of the tentorium, cerebellum or medulla. In the succeeding paper [2] experimental evidence was reported indicating that sudden release of high intracranial fluid tension in dogs causes cellular injury to the central nervous system accompanied by an increased fluid content of the nerve tissues. In the third article [3] the literature in the field was reviewed and the possibility suggested that many of the untoward effects following the rapid drainage of cerebrospinal fluid could in the majority of cases be attributed to shock and edema of the central nervous system rather than to gross displacement of the intracranial contents. This concept was based in great part on a preliminary survey of data obtained in a clinical study of cerebrospinal hydrodynamics in over 200 patients under controlled experimental conditions. It is the purpose of the present communication to report the results of this investigation in greater detail, and to draw such other conclusions as seem warranted by a statistical analysis of the experimental observations.

MATERIAL AND METHODS

In general, the material was drawn from patients who required rachicentesis for diagnostic or therapeutic purposes. There was a large percentage with dementia paralytica (112 of a total of 284 cases),

From the wards and laboratories of the Baltimore City Hospitals, Psychopathic Hospital.

1. Masserman, J. H., and Schaller, W. F.: Intracranial Hydrodynamics: I. Experiments in Human Cadavers, Arch. Neurol. & Psychiat. **29**:1222 (June) 1933.

2. Masserman, J. H., and Schaller, W. F.: Intracranial Hydrodynamics: II. Influence of Rapid Decompression of the Ventriculosubarachnoid Spaces on the Occurrence of Edema of the Brain, Arch. Neurol. & Psychiat. **30**:107 (July) 1933.

3. Masserman, J. H.: Intracranial Hydrodynamics: III. The Clinical Effects of Rapid Drainage of Cerebrospinal Fluid, J. Nerv. & Ment. Dis., to be published.

but the data obtained in these were controlled by results obtained in a series of patients with no demonstrable disease of the central nervous system. The latter group will be known in this article as the "control series."

1. In preparation for the experimental drainage of spinal fluid the patient was given 10 grains (0.5 Gm.) of barbital, and the bowels and bladder were emptied. He was then put at rest in bed for one hour, after which the pulse rate and blood pressure were determined.

2. Following this the patient was placed in a comfortable lateral position, special care being taken to prevent pressure on or constriction of the neck. Lumbar puncture was then performed under local anesthesia by the insertion of an 18 gage needle between the spines of the fourth and fifth lumbar vertebrae.

3. The manometric apparatus employed in most cases was assembled as follows: A 50 cc. glass syringe was equipped with a two-way valve, one arm of which was connected through a rubber U-tube to a 2 mm. bore glass manometer filled to the 100 mm. mark with Ringer's solution. After sterilization of the apparatus in toto the manometer was strapped in a vertical position to the patient's back, and the free arm of the valve inserted into the lumbar needle with the loss of as little fluid as possible. With proper positions of the valve the spinal pressure could then be read on the manometer with a negligible displacement of fluid, or fluid could be withdrawn into the syringe at any desired rate, and the resultant changes in spinal pressure noted.

4. The manometric observations began with a control period during which the cerebrospinal fluid pressure was observed about every three minutes for from twenty to sixty minutes, or until a fairly stable base line had been obtained. No correction for the capillarity of our manometers [4] was deemed necessary, since readings were taken only when the meniscus showed sufficient movement in response to the patient's pulse and respiration practically to negate any slight capillarity the 2 mm. bore manometers might possess.

5. At the end of the control period from 10 to 60 cc. of spinal fluid was removed, all drainages being done if possible within an arbitrarily adopted interval of two minutes. The pressure of the spinal fluid after the drainage was then read every minute until it assumed a fairly stable lower level.

6. Fifteen minutes after the drainage the pulse rate and blood pressure were redetermined, and any subjective symptoms during this interval were recorded. The patient was then allowed to rest on his side until the spinal fluid pressure regained its original level. At this time the pulse rate and blood pressure were again determined.

7. The drainage of cerebrospinal fluid was then repeated, exactly the same amount being removed in two minutes as had been drained initially, after which the second fall in the pressure of the fluid was measured.

8. In most cases only two drainages were performed, and the lumbar needle was removed after the second. In about one fifth (56) of the series, however, the needle was left in situ until the pressure of the cerebrospinal fluid returned to its original level two or more times after the successive removals of the same amount of spinal fluid.

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4. Fay, T.: Mental Deterioration and Deficiency, Am. J. Psychiat. **12**:893 (March) 1933.

9. The intervals of time required for the spontaneous restoration of the pressure were noted in each instance, as were the blood pressure, pulse rate and clinical symptoms of the patient at the time of each drainage.

10. The samples of spinal fluid removed during the repeated drainages were analyzed and compared.

11. Records of the patient's symptoms, pulse rate, blood pressure and objective neurologic examinations were made as indicated during the next forty-eight hours.

12. In many cases spinal punctures were done at approximately eight and twenty-four hours after the original drainage in order to determine the level of the patient's cerebrospinal fluid pressure at these intervals.

This protocol was followed in the majority of the experimental drainages. In certain cases, however, some of the steps were necessarily omitted; in others, additional drainages, tiltings of the patient, reinjections of spinal fluid, spinal injections of modified Ringer's solution, intravenous injections of different concentrations of dextrose and various other procedures were carried out to furnish control or complementary data. These additional observations will be discussed in their proper connection in this and subsequent reports.

NOTATION OF DATA

For convenience in reporting the data, and also to facilitate subsequent discussion, the following symbols will be used:

The initial (original) cerebrospinal fluid pressure of a patient under basic conditions of relaxation will be designated CP_0. The amount of spinal fluid removed in any one drainage—theoretically, the diminution in volume of the ventriculosubarachnoid spaces effected by the drainage—will be called dV, and the decrease in cerebrospinal fluid pressure effected by the first drainage of dV will be designated dP_1. The quotient dV/dP will then represent the amount of fluid withdrawn per unit drop in pressure—a quantity to be known in these studies as the "pressure index."

The time required for the spontaneous restoration of the cerebrospinal fluid pressure to its original level (CP_0) after the first drainage of fluid will be called Tr_1; dP_2 will then signify the amount of decrease in cerebrospinal fluid pressure produced by the second removal of dV, and Tr_2 the time required for the second spontaneous restoration of pressure to CP_0. Similarly, BP_0 and P_0 will denote, respectively, a patient's control blood pressure and pulse rate as taken under basic conditions at the beginning of an experiment, whereas BP_1 and P_1 will represent these quantities as determined fifteen minutes after the first drainage of spinal fluid, BP_2 and P_2 the quantities at the time of the second drainage, etc. All readings of spinal pressure will be given in millimeters of Ringer's solution, and all intervals of time will be expressed in minutes.

STATISTICAL TECHNIC

The mean of a series of observations will be reported with its probable error (P.E.) and standard deviation (σ) whenever the determination of these constants seems of value. The difference between two comparable means (even when the data are correlated) will be considered statistically significant [5] only when its

5. (*a*) Fisher, R. A.: Statistical Methods for Research Workers, London, Oliver & Boyd, 1928. (*b*) Pearl, Raymond: Medical Biometry and Statistics, ed. 2, Philadelphia, W. B. Saunders Company, 1930.

critical ratio exceeds 3.5, and then only if the number of observations (N) in the series is sufficiently large. In this respect it will be noted that the statistical constants for certain of the series are not recorded in the tables or text; the data in these series, in my opinion, were not sufficiently numerous or homogeneous to render their analysis significant. Certain inferences, nevertheless, could conservatively be drawn even from the smaller series, and these inferences together with their necessary reservations are recorded in the text.

CONTROL OBSERVATIONS

The manometric studies early demonstrated that the pressure of the cerebrospinal fluid is highly sensitive to physiologic change, and under normal conditions responds promptly not only to compression of the veins of the neck but also to almost any gross body strain or movement.

For instance, a cough caused a temporary rise in pressure of from 25 to 200 mm. of Ringer's solution, and a violent sneeze sometimes elevated it as much as 280 mm. Conversely, prolonged crying reduced the pressure 10 and 23 mm., respectively, below basic level in two female patients, possibly because of the hyperpneic effect of the sobbing.[6] Bodily conditions not as easily recognized, moreover, also had important effects. Thus, the removal of even the gentle pressure of a pillow on the patient's neck or the correction of a strained or hyperflexed position frequently resulted in substantial decreases in his spinal subarachnoid pressure, and reductions of from 10 to 28 mm. were noted when a patient was allowed to empty the bladder. Psychogenic tension seemed to have almost as great an influence; the cerebrospinal fluid pressure of a patient outwardly quiet but none the less obviously apprehensive was observed to drop 18 mm. after he was assured that the observations were nearly over.

Consideration of these preliminary data rendered it obvious that readings of basic cerebrospinal fluid pressure were reliable only when the patient was under conditions of fairly complete mental and physical relaxation. The experimental technic was therefore designed with these ends in view. The preliminary administration of barbital served to reduce anxiety and apprehension, whereas the repeated infiltration of the site of the puncture with a 0.5 per cent solution of procaine hydrochloride and the employment of a gentle puncture technic rendered the rachicentesis itself almost painless. With the aid of a special type of supportive pillow and the use of appropriate suggestion the patient could then be kept in bed with a needle in his spine for from two to six hours of continuous observation and remain fairly comfortable and cooperative throughout the period. In fact the subjects frequently fell asleep—a circumstance that caused no significant change in cerebrospinal fluid pressure if relaxation had previously been nearly complete.

BASIC CEREBROSPINAL FLUID PRESSURE

The pressure of the cerebrospinal fluid in 22 patients used as controls was observed at five minute intervals during periods of from two to five and one-half hours. Six of the patients did not cooperate to a satisfactory degree; in these the

6. Wolff, H. G., and Lennox, W. G.: Arch. Neurol. & Psychiat. **23**:1097 (June) 1930. Cobb, S., and Fremont-Smith, F.: Cerebral Circulation, ibid. **26**: 734, 1931.

spinal pressure remained relatively constant only during periods of complete relaxation. In ten of the cooperative patients the pressure did not vary more than 14 mm. from its original level during the period of observation. In the six others, slow, steady, unexplained variations in pressure of from 20 to 38 mm. above the respective base-line reading were observed. In no case in which relaxation was fairly complete, however, did sudden fluctuations in pressure such as would invalidate the subsequent experimental hydrodynamic studies occur. It was concluded from these observations, therefore, that under conditions of complete mental and physical relaxation the cerebrospinal fluid pressure either remains relatively stable or is subject to such slow variations (in response to undetermined physiologic changes) that the immediate reductions in pressure during the rapid removal of spinal fluid could be attributed, for practical purposes, solely to the effect of the drainage.

BASIC CEREBROSPINAL FLUID PRESSURE IN VARIOUS POSITIONS

In the total number of cases used for this study the grand mean initial cerebrospinal fluid pressure (CP_0) in the lying position was 147.71 ± 1.89 mm. of water; individual observations ranged between apparently normal extremes of 29 and 224 mm. It is interesting to note (table 1, series 6) that there was no significant difference between the mean CP_0 of patients with dementia paralytica and that of patients with no demonstrable organic disease of the central nervous system; further, this correspondence of CP_0 in the two series is advanced nearer certainty by the fact that there was no significant mean age difference between them (table 1, series 6a). Similar comparisons revealed no significant differences among the respective mean values of CP_0 for male, female, colored and white patients, and therefore rendered the computation of a grand mean CP_0 for the entire series justifiable.

The mean basic pressure of 93 patients in the sitting position was determined to be approximately 397.5 ± 4.21 mm. (table 1, series 1 and 2). Again there was no significant difference in CP_0 between the males and females in the subseries (series 1 and 2)—a finding that strengthens the corresponding observation in the horizontal position. It must be noted, however, that the spinal fluid pressure of a patient in the sitting position varies considerably with his posture and the position of his head [7]—a circumstance that is reflected in the greater probable error of the mean CP_0 of the sitting series as compared with that for the horizontal position.

EFFECTS OF THE RAPID REMOVAL OF SPINAL FLUID

FALL OF CEREBROSPINAL FLUID PRESSURE ON FIRST DRAINAGE (dP_1)

The mean decrements in cerebrospinal fluid pressure resulting from the drainage of various amounts of spinal fluid are listed in table 2 and plotted in the accompanying graph as curve dP_1. It may be seen that the initial drainage of 10 cc. of fluid produces a comparatively greater fall in pressure than the subsequent removal of larger amounts; this circumstance suggests the existence of a physiologic mechanism which protects the central nervous system from too great a diminution in subarachnoid pressure, such as would result from the rapid removal of excessive amounts of cerebrospinal fluid. It must be noted, however, that the

7. Klessens, J.: Changes in Pressure in Lumbar Canal Under Influence of Mechanical Conditions, Nederl. tijdschr. v. geneesk. **1**:1998 (May 15) 1926; abstr., J. A. M. A. **87**:290 (July 24) 1926.

TABLE 1.—*The Initial Pressure of the Cerebrospinal Fluid in Various Series* *

Series No.	Function	Series	Position of Patient	N	Mean ± P.E.†	± P.E.†	Series Compared	Mean Difference ± P.E.†	Critical Ratio	Statistical Conclusions
1	CP_o	Males	S	53	395.35 ± 5.86	63.5 ± 4.15	.....	.....	....	No significant difference between the means of the initial pressure of the cerebrospinal fluid (CP_o) respectively of the male and female series in the sitting (S) position
2	CP_o	Females	S	40	401.50 ± 5.90	55.4 ± 4.23	1 + 2	6.15 ± 8.31	0.74	
1 + 2	CP_o	Total	S	93	397.59 ± 4.21	60.0 ± 2.96				
3a	CP_o	White females	H	77	141.64 ± 2.72	35.4 ± 1.92	.....	.....	....	No significant difference between CP_o of white and colored females in the horizontal (H) position
3b	CP_o	Colored females	H	58	147.70 ± 3.61	40.8 ± 2.56	3a + 3b	6.06 ± 4.74	1.27	
3a + 3b	CP_o	All females	H	135	144.41 ± 2.17	37.5 ± 1.53				
4a	CP_o	White males	H	78	152.15 ± 4.0	52.4 ± 2.82	.....	.....	....	No significant difference in CP_o between white and colored males in the horizontal position; no significant difference in CP_o between males and females in the horizontal position
4b	CP_o	Colored males	H	71	149.52 ± 4.7	59.0 ± 3.32	4a + 4b	2.63 ± 6.18	0.42	
4a + 4b	CP_o	All males	H	149	150.9 ± 3.0	54.5 ± 2.11	3 + 4	6.49 ± 3.70	1.74	
3a + 4a	CP_o	White patients	H	155	147.20 ± 2.36	43.6 ± 1.64	.....	.....	....	No significant difference in CP_o between colored and white patients in the horizontal position
3b + 4b	CP_o	Colored patients	H	129	148.06 ± 3.06	51.5 ± 2.13	3a + 4a and 3b + 4b	0.86 ± 3.81	0.22	
5	CP_o	Controls	H	114	144.2 ± 2.66	42.2 ± 1.88	.....	.....	....	No significant difference in CP_o between controls and patients with dementia paralytica in the horizontal position
6	CP_o	Patients with dementia paralytica	H	112	146.8 ± 2.90	45.8 ± 2.05	5 + 6	2.6 ± 3.94	0.66	
5a	Age	Controls	..	...	40.6 ± 1.28 yrs.	.....	.....	.....	....	No significant difference in the mean ages of the patients in the control series and those with dementia paralytica
6a	Age	Patients with dementia paralytica	..	...	41.5 ± 1.61 yrs.	.....	.....	0.9 ± 2.06	0.43	
3 + 4	CP_o	Grand mean	H	284	147.71 ± 1.89	47.3 ± 1.34	3 + 4 and 1 + 2	249.7 ± 4.6	54.4	CP_o in the sitting position = 2.9 times CP_o in the lying position

* In this and tables 2, 3 and 4 the following abbreviations are used: H, horizontal (lying) position; S, sitting position; csf., cerebrospinal fluid; csfp., cerebrospinal fluid pressure. The other abbreviations will be found on page 525 of the article.

† In millimeters of Ringer's solution.

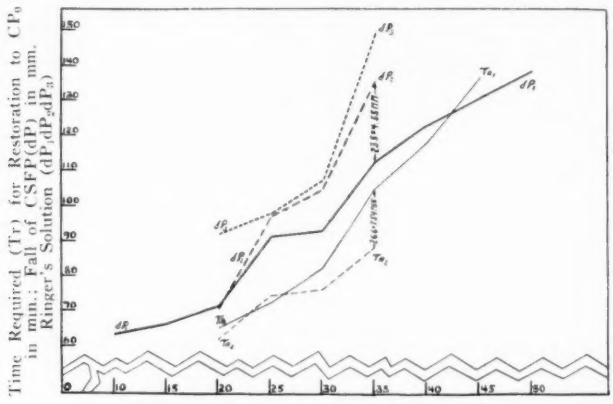
† In millimeters of Ringer's solution.

TABLE 2.—*The Fall in Spinal Subarachnoid Pressure After the First, Second and Third Drainage of Various Amounts of Cerebrospinal Fluid*

Series No.	Function	dV, Cc.	Series	Position	N	Mean ± P.E.*	σ ± P.E.*	dV/dP	Series Compared	Mean Difference ± P.E.*	Critical Ratio	Statistical Conclusions
1	dP_1	10	All patients	H	35	63.3 ± 2.02	17.75 ± 1.43	0.159				
2	dP_1	15	All patients	H	18	66.4 ± 3.78	23.80 ± 2.68	0.226				
3	dP_1	20	All patients	H	41	71.7 ± 2.82	26.9 ± 2.0	0.278	.....	.....	....	No significant difference between dP_1 and dP_2 for drainages of 20 cc. of spinal fluid
4	dP_2	20	All patients	H	15	70.9 ± 4.80	27.6 ± 3.39	0.272	3 + 4	0.8 ± 5.56	0.14	
5	dP_3	20	All patients	H	11	92.5 ± 6.15	30.2 ± 4.35	0.216				
6	dP_1	20	All patients	S	46	126.6 ± 4.77	47.2 ± 3.38	0.159	3 + 6	54.9 ± 2.74	20.0	dP_1 for 20 cc. in sitting position is 1.8 times dP_1 in horizontal position
7	dP_1	25	All patients	H	33	91.9 ± 4.67	39.7 ± 3.3	0.272	.....	.....	....	No significant difference between dP_1 and dP_2 for dV = 25 cc. of fluid
8	dP_2	25	All patients	H	19	97.8 ± 6.61	42.8 ± 4.67	0.256	7 + 8	5.9 ± 8.09	0.73	
9	dP_3	25	All patients	H	14	98.2 ± 6.28	34.8 ± 4.44	0.254				
10	dP_1	30	All patients	H	15	93.2 ± 4.14	39.7 ± 2.92	0.321	.....	.....	....	No significant difference between dP_1 and dP_2 for dV = 30 cc. of fluid
11	dP_2	30	All patients	H	42	105.1 ± 5.44	31.3 ± 3.85	0.285	10 + 11	11.9 ± 6.84	1.72	
12	dP_3	30	All patients	H	11	103.2 ± 5.82	28.7 ± 4.12	0.279				
13a	dP_1	35	Female patients	H	33	107.7 ± 4.44	37.8 ± 3.14	0.325	.....	.....	....	No significant difference in dP_1 for dV = 35 cc. between male and female patients
13b	dP_1	35	Male patients	H	35	117.3 ± 4.46	39.1 ± 3.15	0.299	13a + 13b	9.6 ± 6.32	1.50	
13	dP_1	35	All patients	H	68	112.7 ± 3.18	38.9 ± 2.24	0.311				
14	dP_2	35	All patients	H	41	136.2 ± 3.25	30.8 ± 2.29	0.265	13 + 14	23.5 ± 4.55	5.15	Grand mean dP_2 for dV = 35 cc. exceeds dP_1 for the same amount by 23.5 mm. of Ringer's solution
15	dP_3	35	All patients	H	20	150.0 ± 6.40	42.5 ± 4.53	0.233	14 + 15	13.8 ± 7.18	1.92	
16	dP_1	40	All patients	H	19	123.4	.....	0.325	.....			Data not sufficiently numerous for significant statistical analysis
17	dP_2	40	All patients	H	5	134.3	.....	0.296	.....			
18	dP_1	45	All patients	H	12	131.5	.....	0.341	.....			
19	dP_2	45	All patients	H	5	144.7	.....	0.310	.....			
20	dP_1	50	All patients	H	8	139.5	.....	0.356	.....			
21	dP_2	50	All patients	H	4	142.8	.....	0.349	.....			

* In millimeters of Ringer's solution.

disparity of the pressure index for various amounts of dV does not hold true in an individual case when the spinal fluid is drained more slowly. This was illustrated in a series of patients, from each of whom from 40 to 60 cc. of fluid was drained in successive amounts of 10 cc. each, time being allowed between drainages



Number of cc. (dV) of Spinal Fluid Withdrawn

The mean intervals of time required for the restoration of the initial cerebrospinal fluid pressure after the drainage of various amounts of cerebrospinal fluid, and the mean decreases of cerebrospinal fluid pressure effected by these drainages. (See text, pp. 3, for key to symbols).

TABLE 3.—*The Time Required (Tr) for Spontaneous Restoration of the Cerebrospinal Fluid Pressure to Its Initial Level After the First and Second Drainages*

Series No.	Function	dV, Cc.	N	Mean (± P.E.) Minutes	dV/Tr, Cc./Min.	Conclusions
1	Tr_1	20	13	65.5	0.305	
2	Tr_2	20	10	62.0	0.322	
3	Tr_1	25	15	72.7	0.342	
4	Tr_2	25	12	74.8	0.336	
5	Tr_1	30	15	82.7	0.361	
6	Tr_2	30	10	76.7	0.396	The time required for the restoration of cerebrospinal fluid pressure to its original level is significantly less (−26.6 ± 7.54 minutes) after the second drainage of 35 cc. of spinal fluid than after the first
7	Tr_1	35	42	115.4 ± 4.05	0.305	
8	Tr_2	35	24	88.8 ± 6.36	0.396	
9	Tr_1	40	16	118.8	0.322	
10	Tr_1	45	12	136.5	0.346	
11	Tr_1	50	6	154.6	0.318	
1-3-5-7	Aver. dV/Tr_1	20-35	85	.....	0.319	
2-4-6-8	Aver. dV/Tr_2	20-35	56	.....	0.376	Difference = +0.057 cc. per min.

for complete readjustments of the pressure in the ventriculosubarachnoid system.[8] The manometric studies in this series indicated that whereas the drainage of the

8. Masserman, J. H.: Cerebrospinal Hydrodynamics: V. The Coefficient of Volume Elasticity of the Human Ventriculosubarachnoid System, to be published.

first 10 cc. of spinal fluid causes a slightly greater fall in cerebrospinal fluid pressure, the slow removal of subsequent amounts of 10 cc. results in almost constant values of dV/dP until the total drainage exceeds 50 cc., when further readings may well be rendered inaccurate by partial collapse of the spinal subarachnoid spaces. It appears, therefore, that whereas the pressure index in contrasted series of patients seems to show a compensatory increase in value as progressively larger amounts of fluid are rapidly removed, the index nevertheless remains fairly stable in the same person for the gradual drainage of small amounts of fluid.

TIME REQUIRED FOR FIRST SPONTANEOUS RESTORATION OF CEREBROSPINAL FLUID PRESSURE (Tr_1)

The time required for the spontaneous restoration of the cerebrospinal fluid to its original level after the first drainage of fluid is tabulated in table 3, and plotted as curve Tr_1 in the graph. As may be seen, the mean time for the restoration of the cerebrospinal fluid pressure to basic level varies from about an hour after the drainage of 20 cc. of spinal fluid to about two and one half hours after the drainage of 50 cc. The data, however, are most complete for the drainage of 35 cc. (table 3, series 7), for which the mean Tr_1 is 115.4 ± 4.05 minutes, with a standard deviation of 39 minutes. These figures, of course, are of statistical value only, since the actual Tr_1 after the drainage of 35 cc. of spinal fluid in any particular case may range from a half hour to longer than three hours. In only 3 cases of the entire series, however, did the fluid pressure fail to restore itself to normal within the latter interval.

In nearly all cases the curves of recovery of pressure were comparatively steep immediately after the drainage and flatter as the initial level of cerebrospinal fluid pressure was approached. This observation may indicate that the vascular mechanism of maintaining cerebrospinal hydrodynamic equilibrium is probably most active immediately after the drainage of fluid, whereas the comparatively slow secretion of cerebrospinal fluid later completes the cycle of the restoration of pressure.

COURSE OF THE CEREBROSPINAL FLUID PRESSURE AFTER RESTORATION TO BASIC LEVEL

The curve of the cerebrospinal fluid pressure after its restoration to CP_0 was observed in 41 patients in whom the lumbar manometric apparatus was left in situ, and spinal pressures were read every five minutes for from one to three hours after the expiration of Tr_1. The data so obtained do not lend themselves to compact tabulation, but may be grouped in this manner:

In 18 cases in which less than 20 cc. of spinal fluid had been drained the cerebrospinal fluid pressure after regaining CP_0 either remained about constant (7 cases) or continued to rise from 5 to 14 mm. higher. In 23 cases in which more than 30 cc. had been rapidly drained the rise above CP_0 was more marked and constant; in these instances the cerebrospinal fluid pressure mounted from 12 to 108 mm. above the base-line in the forty to one hundred and forty-five minutes following the expiration of Tr_1.[9] In general, the higher and more prompt rises were observed in cases in which more than 40 cc. of spinal fluid had been aspirated. In the latter instances, also, the cerebrospinal fluid pressure fell but little from

9. Balduzzi (The Pressure of the Cerebrospinal Fluid After Lumbar Puncture, Rev. neurol. **1**:1036, 1927) reported a similar finding for drainages of as little as from 5 to 15 cc. of spinal fluid.

its zenith during the subsequently necessarily short period (from thirty to seventy minutes) of continuous observation, while a greater descent toward CP_0 was observed in cases in which only 30 cc. had been drained.

In order to determine the course of the cerebrospinal fluid pressure at longer intervals following the initial spinal drainage, 32 patients were repunctured approximately at eight and again at twenty-four hours after the first removal of fluid. In those cases in which 20 cc. or less had been drained the average pressures at eight and twenty-four hours showed no significant changes from the mean CP_0 of the series. In 19 cases in which more than 30 cc. had been removed initially, the average pressure at eight hours was still approximately 23 mm. above normal, although 3 showed pressures about equal to CP_0, and 4 others gave readings from 5 to 12 mm. below the base level. The series included only 13 patients from whom from 30 to 50 cc. had been withdrawn and who had been repunctured at the end of twenty-four hours; in these, the readings of cerebrospinal fluid pressure at the twenty-four hour interval varied so widely in both directions (minus 18 to plus 36 mm.) from the initial pressure in each case that no definite inferences could be drawn. In general, however, the conclusion seems justified that following the drainage of from 30 to 50 cc. of spinal fluid its pressure returns to normal in an average time of about two hours, and the pressure then mounts to a higher level, which in the majority of cases is maintained for at least eight hours, whether or not the puncture needle is left in situ to "plug the hole in the dura."[10] The determination of the subsequent course of the pressure curve must await further data.

EFFECT OF SECOND DRAINAGE OF CEREBROSPINAL FLUID ON ITS PRESSURE (dP_2)

If, after the restoration of the cerebrospinal fluid to its basic level the upward trend of the pressure curve is interrupted by repeating the drainage of the original (dV) amount of spinal fluid, the extent of the second drop (dP_2) in pressure so produced has a certain theoretical significance. The mean values of this quantity for increasing amounts of dV are tabulated in table 2 and plotted as curve dP_2 in the graph. Although it is noteworthy that curves dP_1 and dP_2 in general diverge between the ordinates of 20 and 35 cc. dV, the data may be considered as approaching determinative value only for the drainage of the latter amount of fluid. For this quantity, however, the difference between the means of dP_1 and dP_2 (table 2, series 14) is statistically significant. In other words, the second rapid removal of 35 cc. of spinal fluid after the spontaneous restoration of the subarachnoid pressure to normal produces, in the great majority of cases, a significantly greater (23.5 ± 4.55 mm.) fall of cerebrospinal fluid pressure than did the first drainage of the same amount of fluid. The possible interpretation of this interesting phenomenon will be discussed.

TIME REQUIRED FOR SECOND RESTORATION OF CEREBROSPINAL FLUID PRESSURE (Tr_2)

The data are here again statistically determinative only for the drainage of 35 cc. of fluid; for this dV, Tr_1 is about twenty-six minutes longer than Tr_2 (table 3, series 8), and the mean difference is about three and a half times its

10. The theory of MacRobert (The Cause of Lumbar Puncture Headache, J. A. M. A. **70**:1350 [May 11] 1918) that leakage from the puncture hole causes hypotension of the cerebrospinal fluid and postpuncture headache is not supported by my experiments.

probable error. One obvious interpretation of this finding is that the second drainage of 35 cc. of spinal fluid causes a greater (and/or summated?) stimulus to the production of the fluid than did the first drainage. However, several other factors may play a rôle in determining the intervals of time required for the spontaneous restoration of CP_0 after the successive drainages of various amounts of spinal fluid; these factors also will be commented on subsequently in this paper.

DRAINAGES AND INTERVALS OF RESTORATION SUBSEQUENT TO Tr_2

The values of dP_3 (the extent of fall in pressure during the third drainage of cerebrospinal fluid) were determined in 56 cases, but the data are so distributed among the various series that only a general inference may be drawn; viz., dP_3 becomes progressively greater than dP_2 (table 2 and the graph) as the amount of dV increases. The largest series occurs for the third drainage of 35 cc. of fluid (table 2, series 15), but even here the difference between dP_2 and dP_3 is not statistically significant. Nevertheless, the shape and position of curve dP_3 indicate that the third removal of spinal fluid, in general, produces greater falls in cerebrospinal fluid pressure than did either of the first two drainages, especially for large amounts of dV.

Measurements of the intervals of restoration and the fall in the cerebrospinal fluid pressure produced by the drainages of fluid subsequent to dP_3 were made in only 12 instances, and for clinical reasons to be discussed were restricted to cases in which comparatively small amounts of spinal fluid were repeatedly removed. The data are here again difficult to summarize; however, 3 cases from the series may be cited as fairly typical:

CASE 1.—A white man, aged 28, who was mentally deficient, showed: BP_0, 108 systolic and 72 diastolic; P_0, 72; CP_0, 208 mm.; dV, 20 cc.; dP_1, 60 mm.; Tr_1, thirty minutes; dP_2, 66 mm.; Tr_2, forty minutes; dP_3, 62 mm.; Tr_3, forty minutes; dP_4, 68 mm.; Tr_4, twenty-five minutes; dP_5, 71 mm.; Tr_5, forty-five minutes, and dP_6, 62 mm. Fifteen minutes after dP_6 the blood pressure was 104 systolic and 78 diastolic, and the pulse rate, 72. There were no complaints of discomfort.

CASE 2.—A colored woman, aged 38, a psychopathic person, showed: BP_0, 128 systolic and 84 diastolic; P_0, 82; CP_0, 173 mm.; dV, 30 cc.; dP_1, 98 mm.; Tr_1, 92 min.; dP_2, 118 mm.; Tr_2, sixty minutes; dP_3, 146 mm.; Tr_3, eighty minutes, dP_4, 150 mm. She complained of headache and backache. At the conclusion of the tests the blood pressure was 132 systolic and 88 diastolic and the pulse rate 82. Thirty cubic centimeters of spinal fluid was reinjected into the spinal canal, after which the cerebrospinal fluid pressure rose to 212 mm. No change in symptoms was produced by the reinjection of the spinal fluid. Partial relief followed the administration of 250 cc. of 20 per cent dextrose solution intravenously.

CASE 3.—A colored woman, aged 34, with dementia paralytica, showed: BP_0, 112 systolic and 68 diastolic; P_0, 70; CP_0, 155 mm.; dV, 35 cc.; dP_1, 102 mm.; Tr_1, one hundred and five minutes; dP_2, 128 mm.; Tr_2, sixty minutes; dP_3, 142 mm.; Tr_3, eighty minutes; dP_4, 149 mm. Thirty-five cubic centimeters of spinal fluid was reinjected, after which the cerebrospinal fluid pressure rose to 194; the blood pressure to 110 systolic and 68 diastolic, and the pulse rate to 72. She complained of slight headache.

It is evident that the results, especially those obtained on the repetition of the lesser drainages, vary within wide limits, illustrating the necessity for a large series of observations before significant analyses of data can be carried out. How-

ever, it was observed that Tr intervals subsequent to Tr_2 increased in length in 9 of the 12 cases of the series, indicating that the stimulative effect of drainage of spinal fluid on its production becomes progressively less effective after the second removal of fluid. It is also interesting to note that in 4 cases approximately 100 cc. of spinal fluid could be drained in divided amounts during a three hour period without producing a spinal fluid pressure lower than the initial level at the end of this interval.

REMOVAL AND REINJECTION OF SPINAL FLUID

In order to determine whether the effects of pressure produced by removal of cerebrospinal fluid were reversible, various amounts of spinal fluid were aspirated and then reinjected, each operation taking one minute, the drainage and reinjection being separated by intervals of two minutes during which the changes of pressure in the cerebrospinal fluid were noted. It was found that repeated drainages and reinjections of even small amounts of fluid produced progressive increases in the tension of the cerebrospinal fluid, so that the oscillations of pressure plotted from any of the experiments assumed the form of continuous reversed spikes about an abscissal axis that sloped rather steeply upward. This again may perhaps best be illustrated by citing a typical case:

CASE 4.—A colored woman, aged 24, a psychopathic personality, showed: BP_0, 84 systolic and 60 diastolic; P_0, 78, and CP_0, 63 mm. Ten cubic centimeters of spinal fluid was aspirated in one minute, after which the cerebrospinal fluid pressure fell to 6 mm.; 10 cc. was reinjected and the cerebrospinal fluid pressure rose to 82 mm. These procedures were repeated, and the following readings were obtained: 10 cc. aspirated, 9 mm., reinjected, 82 mm.; 10 cc. aspirated, 34 mm., reinjected, 102 mm.; 10 cc. aspirated, 38 mm., reinjected, 112 mm. This cerebrospinal fluid pressure was maintained for fifteen minutes. Then 10 cc. more was removed; the pressure dropped to 32 mm.; 10 cc. was reinjected, and the pressure rose to 120 mm. The blood pressure about forty minutes after beginning the experiment was 98 systolic and 74 diastolic, and the pulse rate was 92. Severe backache and mild nausea were present for twenty-four hours, and were only slightly relieved by the intravenous injection of 250 cc. of a 20 per cent dextrose solution.

When amounts larger than 10 cc. of spinal fluid were alternately drained and reinjected, even more striking increases in the cerebrospinal fluid pressure were produced. Thus, in 8 cases in which approximately 40 cc. was aspirated the mean first reduction in the cerebrospinal fluid pressure was 117.5 mm.and the rise above the initial level of the spinal fluid pressure on reinjection was 31.2 mm., whereas the second aspiration produced a mean fall of 138.5 mm. and the second reinjection a rise of 81.0 mm. above CP_0. In 4 of the patients puncture was done again approximately four hours after the initial drainage. Since the results in the 4 were similar, only 1 illustrative protocol need be cited:

CASE 5.—A white man, aged 32, with schizophrenia, showed: BP_o, 110 systolic and 65 diastolic, and the pulse rate, 72. CP_o, at 10:35 a. m. was 85 mm. Forty cubic centimeters of cerebrospinal fluid was aspirated in three minutes; the cerebrospinal fluid pressure fell to minus 10 mm. The fluid was reinjected in three minutes; the pressure was 110. Forty cubic centimeters was withdrawn; the pressure became plus 14; the fluid was reinjected and the pressure became 142; fluid was again withdrawn and the pressure became 26. At 2:30 p. m., the patient complained of severe pain in the head, back and legs; the blood pressure was 120 systolic and 88 diastolic, and the pulse rate was 60. Spinal puncture was repeated; the cerebrospinal fluid pressure was 115. Forty cubic centimeters were removed in four minutes; the pressure dropped to 45 mm. below the spinal level. The headache was somewhat relieved, but returned in about an hour. The patient then was given 250 cc. of 20 per cent dextrose solution intravenously. The headache and pains in the legs were relieved about thirty minutes after the injection.

EFFECT OF RAPID DRAINAGE OF SPINAL FLUID ON PERIPHERAL ARTERIAL BLOOD PRESSURE AND PULSE RATE

Table 4 reveals that the only significant changes in blood pressure occurring in any of the series were: (1) a probable decrease of about 9 mm. of mercury in the systolic blood pressure approximately fifteen minutes after the rapid drainage of 20 cc. of spinal fluid with the patient in the sitting position (table 4, series 7); (2) a probable increase in the systolic pressure of about 7 mm. and a corresponding increase in the diastolic pressure of 9 mm. occurring from one to three hours after the removal of from 30 to 50 cc. of spinal fluid from patients in the horizontal (lying) position (table 4, series 5).

It may, of course, be contended that these experimental deviations are of small amount, that the critical ratios are barely significant (from 3.54 to 4.75) and that even this significance may be vitiated by the inaccuracies inherent in clinical readings of blood pressure. However, it was to overcome just such criticisms that the statistical technic here employed was adopted. Thus, only the differences in a single person (table 4, series 5 to 8) between the control blood pressure and the blood pressure under investigation were recorded—an accurate method for determining the range of deviation of the differences between correlated variables. Further, the data were so entered in the tables of distribution that a difference in readings of blood pressure of 5 mm. of mercury in either direction from the control observation was classified as zero deviation. As to the possible variability of the blood pressure readings, it must be considered that unavoidable inaccuracies in a series of measurements do not materially affect their statistical constants [5b] if only the series is sufficiently large and the data are unprejudiced. Finally, when it is remembered that a mean difference only three and a half times its probable error still stands a better than 50:1 chance of being significant, it becomes evident that inferences based on such statistical data are at least worthy of record and further investigation.

As to the effect of the rapid drainage of spinal fluid on the pulse rate, the observations may be summarized briefly: No significant changes in pulse rate directly attributable to the drainage of spinal fluid were observed in any of the series.

TABLE 4.—*The Changes in Blood Pressure After the Rapid Drainage of Various Amounts of Cerebrospinal Fluid*

Series	Function	dV, Cc.	Position	N	Mean ± P.E., Mm. of Hg	σ ± P.E.	Series Compared	Mean Difference ± P.E.	Critical Ratio	Conclusions*
1	BP_0: grand mean initial Systolic	.....	..	188	126.4 ± 1.43	29.21 ± 1.04				
	blood pressure Diastolic				81.1 ± 0.79	15.9 ± 0.55				
2	BP_1: blood pressure fifteen minutes after the drainage of cerebrospinal fluid	10-30	H	45	121.2 ± 3.60	35.8 ± 2.55	1-2	−5.2 ± 3.87	1.31	No significant change in blood pressure after the rapid removal of from 10 to 30 cc. of spinal fluid from patients in the lying position
					84.6 ± 2.38	23.7 ± 1.67		+3.5 ± 2.51	1.39	
3	BP_2: blood pressure at the time of the second drainage of dV	10-30	H	32	130.6 ±3.13	26.2 ± 2.21	1-3	+4.2 ± 3.44	1.22	
					87.4 ± 1.97	16.5 ± 1.39		+5.3 ± 2.12	2.50	
4	BP_1: blood pressure fifteen minutes after drainage of from 35 to 50 cc. of csf.	35-50	H	60	117.0 ± 2.12	24.3 ± 1.49	1-4	−9.4 ± 2.56	3.66	There is a probable fall of 9.4 mm. in systolic blood pressure 15 minutes after the rapid drainage of 35 to 50 cc. from patients in the lying position
					78.7 ± 1.91	22.0 ± 1.35		−2.4 ± 2.07	1.16	
					Mean Difference ± P.E.					
5	(BP_0-BP_2) The difference between the initial B.P. and the B.P. at the second drainage of csf.	35-50	H	32	+7.2 ± 2.03	16.9 ± 1.43	.....		3.54	Probable rise in both systolic (+7.2 mm.)and diastolic (+8.9 mm.) B.P. from 1 to 3 hours after the drainage of from 35 to 50 cc. of csf. in lying position
					+8.9 ± 2.18	18.2 ± 1.54	.....		4.11	
6	(BP_0-BP_3) The difference between the initial B.P. and the B.P. at the third drainage of csf.	35-50	H	21	+5.94 ± 2.21	14.9 ± 1.57	.....		2.69	Rise of diastolic B.P. after the rapid drainage of from 35 to 50 cc. of csf. probably persists from 1 to 3 hours (or longer) after its occurrence, but the series is too small for a general conclusion
					+7.21 ± 2.39	16.3 ± 1.69	.....		3.02	
7	(BP_0-BP_1) The difference between the initial B.P. and the B.P. fifteen minutes after the drainage of csf.	20	S	32	−9.4 ± 1.98	16.65 ± 1.39	.....		4.75	Probable fall in systolic B.P. after the rapid drainage of 20 cc. of csf. from patients in the sitting position
					−1.12 ± 1.35	11.15 ± 0.94	.....		0.83	
8	(BP_0-BP_{4Hr}) The difference between the initial B.P. and the B.P. four hours after the drainage of csf.	20	S	35	−0.53 ± 1.41	12.25 ± 0.99	.....		0.41	No significant change in B.P. 4 hours after the rapid drainage of 20 cc. of spinal fluid from patients in the sitting position
					−0.3 ± 1.05	9.1 ± 0.73	.....		0.29	

* Since, in general, the series in this table are small and the critical ratios low, the inferences drawn from the data presented had best be considered probable rather than determinative.

CLINICAL EFFECT OF RAPID DRAINAGE OF CEREBROSPINAL FLUID

Headache, beginning most frequently about an hour after the drainage and usually localized in the occiput or the nape of the neck, was the most common postpuncture complaint. In the control series headaches occurred with a frequency that began with about 6 per cent for drainage of 15 cc. or less, and rose to 93 per cent (26 of 28 cases) when more than 40 cc. of spinal fluid was removed. Backache, nausea, vertigo, pyrexia and tinnitus followed in order of diminishing frequency in the various series, one or more of these symptoms occurring in about a fifth of the control patients from whom less than 25 cc. of cerebrospinal fluid had been removed, whereas in the group from whom 35 cc. had been derived the percentage rose to 38. Headache, stiffness of the neck and back and pain in the legs (frequently of bilateral sciatic distribution) were almost invariably complained of by patients from whom more than 40 cc. of fluid had been rapidly drained; moreover, about a third of the patients in this series suffered transient rises of rectal temperature of from 1 to 5 F. In 1 patient from whom 60 cc. of spinal fluid had been rapidly aspirated these symptoms were accompanied by paralysis of both lower extremities, the loss of the knee jerk and ankle jerk and hypesthesia to touch and pinprick as high as the first lumbar segmental distribution. The syndrome in this case disappeared gradually over a period of ten days, and left no apparent aftereffects other than inducing extreme caution on my part in conducting subsequent rapid drainages of more than 50 cc. of spinal fluid. Complaints following puncture in the other cases lasted from a few hours to six days, and responded to several forms of treatment, including the intravenous injection of hypertonic solutions of dextrose.[11]

Other interesting analyses of the data were possible. For instance, it was noted that only 6 of 41 patients from whom 20 cc. of spinal fluid had been drained while they were in the horizontal position subsequently complained of headache or backache, whereas these symptoms occurred with about three times this frequency (19 of 46 cases) among patients from whom the same amount of fluid had been withdrawn while in the sitting position. This correlates well with the observation that the drainage of 20 cc. of spinal fluid produces nearly twice as great a decrease in the pressure of the cerebrospinal fluid in a patient in the sitting position as it does in one lying horizontally (table 2, series 3 and 6), and confirms in another respect the etiologic relationship between the rapidity and extent of the disturbance in the pressure of the cerebrospinal fluid on the one hand and the incidence and severity of postrachicentesic symptoms on the other. Again, in cases in which 35 cc. or more of spinal fluid was drained and then reinjected, postpuncture symptoms were universal and severe. Considering that in these cases only the patient's own sterile warm spinal fluid was reintroduced into the ventriculosubarachnoid labyrinth, the increase in postrachicentesic symptoms must be attributed solely to the marked disturbances in the fluid balance of the central nervous system produced during the experiments in reinjection. The postpuncture syndrome in these cases was, in fact, so undeniably much more severe than in the simple experiments in drainage that the series of the former was limited to what seemed to be the minimum significant number.

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11. Masserman, J. H.: Cerebrospinal Hydrodynamics: VII. The Effects of the Intravenous Injection of Hypertonic Solutions of Glucose on Cerebrospinal Hydrodynamics, to be published.

One other comparison is perhaps worthy of mention as being possibly confirmatory of a long standing clinical impression: Postpuncture symptoms were complained of only about a third as frequently in the series of patients with dementia paralytica as in the control series of patients with no organic disease of the central nervous system.[12] This could not be attributed solely to the inability of the patients with dementia paralytica to communicate their complaints, since the postpuncture syndrome in both series was studied only in patients who were comparatively rational and cooperative.

CHANGES IN COMPOSITION OF THE CEREBROSPINAL FLUID

In 54 control cases the composition of the spinal fluid obtained at the first puncture was compared with that of specimens drained from two to forty-eight hours later. In 20 cases in which 15 cc. or less had been drained, no constant changes in the composition of the fluid were noted other than a tendency of specimens subsequent to the first to have a comparatively lower specific gravity (mean difference, -1.6 ± 0.51). Other changes in the cerebrospinal fluid following drainages of between 20 and 35 cc. (19 patients) were inconstant; however, in the 15 cases in which more than 40 cc. was rapidly drained the alterations in the fluid were fairly well marked and consistent. Thus, the reaction to the Pandy test, negative in the initial sample, became doubtfully positive in subsequent samples in 5 cases and definitely positive in 7. In 12 cases a pleocytosis developed, reached its maximum in about twelve hours, and then subsided. In 8 of the 12 cases the fluid contained less than 500 cells per cubic millimeter; in the other 4, counts of 800, 1,250, 1,400 and 2,800 cells per cubic millimeter were recorded. The differential counts for the milder pleocytoses showed about equal numbers of lymphocytes and granulocytes; the higher counts, however, consisted of from 62 to 90 per cent granulocytes, and included considerable numbers of irregularly shaped neutrophilic cells of unidentified origin. Since the pleocytoses with the higher counts were frequently accompanied by severe headaches, radiating pains in the legs, mild fever and suggestive Brudzinski and Kernig signs, the question of septic inoculation of the arachnoid space by the needle puncture naturally arose. However, the possibility that infection had occurred in these cases is lessened by the fact that the spinal fluid in each instance was sterile on culture and by the circumstance that the syndrome disappeared completely within six days in all cases except the instance of paraplegia previously recorded. It may be inferred, therefore, that the changes in the cerebrospinal fluid were probably due to aseptic meningeal irritation produced by the rapid reduction in the pressure of the cerebrospinal fluid. In this connection several reports may be found in the literature relative to sterile pleocytoses following either encephalography[13] or simple spinal drainage.[14]

In patients subjected to drainage and reinjection of more than 40 cc. of spinal fluid, subsequent pleocytosis of the fluid was invariable, and reached counts of 3,600 cells, with granulocytes predominating in most instances. It is therefore

12. A similar observation was noted by Nelson, M. O.: Postpuncture Headaches, Arch. Dermat. & Syph. **21**:615 (April) 1930.

13. Hermann, G.: Med. Klin. **18**:1146, 1922. Friedman, E. D.; Snow, W., and Kasanin, S.: Experiences with Encephalography via the Lumbar Route, Arch. Neurol. & Psychiat. **19**:762 (May) 1928.

14. Hurxthal, L. M.: Sterile Meningitis Following Lumbar Puncture, J. A. M. A. **100**:1489 (May 13) 1933.

interesting to note that alternate reductions and increases of cerebrospinal fluid pressure can alone produce evidence of injury to the central nervous system, such as headaches, pyrexia, signs of meningeal irritation and sterile pleocytosis.

The changes in the composition of the spinal fluid in cases of syphilis of the central nervous system followed in general the changes already outlined, with one interesting addition: In 2 of the 15 cases studied the Wassermann reaction, negative in the first 40 cc. of cerebrospinal fluid drained, became positive in samples removed four hours later. In both cases Wassermann tests of the blood were positive.

MULTIPLE DRAINAGES

In order to check the constancy of the findings in individual cases, 40 patients received spinal punctures at about weekly intervals, the total number of punctures in each case ranging from two to eight. The data gathered in this series unavoidably lack uniformity since, for an individual patient, the number of punctures, the intervals between them and the amount of fluid drained had each to be governed not alone by considerations of research but also by clinical expediency. Nevertheless, certain impressions, partially substantiated by statistical considerations, are worthy of record.

The pressure of the cerebrospinal fluid under basal conditions of the individual patient apparently varies considerably from week to week. In the present series the amount of this variation differed among various patients, and bore no unalterable relationship to the patient's treatment or clinical condition. In 26 of the 40 patients the greatest deviation of cerebrospinal fluid pressure from the average CP_o in each case was 20 mm. or less; in the other 14 patients deviations as high as 62 mm. from the average were encountered. The mean maximum deviation from the average of the entire series (N=40) was 25 ± 1.92 mm.

The amount of decrease in cerebrospinal fluid pressure produced by the drainage of a given amount of fluid—the pressure index—varied much less from week to week in an individual patient than did the CP_o, although comparatively large deviations (one and a half times the average dV/dP) were occasionally encountered. However, dP_1 and dP_2 in this series had the same relationship to each other as has been described for the larger series of single punctures. Conversely, the least stable quantity measured in the series of multiple drainages seemed to be that of the time required for the spontaneous restoration of cerebrospinal pressure—Tr_1. For instance, in the same patient, 20 cc. of spinal fluid would be approximately replaced in one hundred minutes in one experiment, whereas at another drainage several days later only sixty-five minutes would be required, or at a third as long as one hundred and forty minutes. This variability of Tr_1 from drainage to drainage held true for all the experimental quantities of dV. It may be inferred, therefore, that the rate of secretion of spinal fluid in response to its rapid removal varies considerably from week to week. The physiologic circumstances that control this variability must, however, await further specific investigation.

COMMENT

SIGNIFICANCE OF MANOMETRIC READINGS

In an evaluation of the methods of studying cerebrospinal hydrodynamics a fundamental problem naturally arises: Can the fluid pressure in the spinal subarachnoid space be considered a reliable index of the pressures that obtain throughout the ventriculosubarachnoid system? An affirmative answer to this question becomes apparent

when it is considered that the ventriculosubarachnoid-perivascular spaces of the brain and spinal cord form a continuous self-enclosed labyrinth,[15] and that the cerebrospinal fluid can, under normal conditions, flow freely from one portion of its complex container to another.[16] On physical grounds alone, therefore, it follows that in the horizontal position of the patient the pressures in all portions of the ventriculosubarachnoid system are practically equal,[17] whereas in any other position simultaneous manometric determinations in various portions of the system would come to rest in the same horizontal plane.[18] In any case the pressures in all parts of the ventriculo-

15. (*a*) Key, A., and Retzius, A.: Studien in der Anatomie des Nervensystems, Stockholm, P. S. Norstedt & Söner, 1875, vol. 1. (*b*) His, quoted by Aycock, W. L., in Dana, C. L.; Davis, T. K., and others: Human Cerebrospinal Fluid: An Investigation of the Most Recent Advances as Reported by the Association for Research in Nervous and Mental Diseases, (1924, vol. 4, p. 220), New York, Paul B. Hoeber, Inc., 1926. (*c*) Hassin, G. B.: Hydrocephalus, Arch. Neurol & Psychiat. **27**:406 (Feb.) 1932; Histologic Studies in Meningitis, ibid. **28**: 789 (Oct.) 1932; So-Called Circulation of the Cerebrospinal Fluid, J. A. M. A. **101**:962 (Sept. 9) 1933 (additional references on this subject can be found in Masserman,[3] footnotes 67, 68 and 69).

16. Dandy, W. E., and Blackfan, H. D.: An Experimental and Clinical Study of Internal Hydrocephalus, J. A. M. A. **61**:2216 (Dec. 20) 1913; Internal Hydrocephalus, Am. J. Dis. Child. **8**:406 (Dec.) 1914; Internal Hydrocephalus, ibid. **14**: 424 (Dec.) 1917. Haller: Die Mechanik des Liquor cerebrospinalis Mitteil, Abhandl. d. k. Grenzgeb. d. Med. u. Chir. **30**:472, 1918. Ayer, J. B.: Spinal Subarachnoid Block as Determined by Combined Cisternal and Lumbar Punctures, Arch. Neurol. & Psychiat. **7**:38 (Jan.) 1922; Cerebrospinal Fluid Pressure from the Clinical Point of View, ibid. **14**:440 (Oct.) 1925. Solomon, H. C.; Thompson, L. J., and Pfeiffer, H. M.: Circulation of Phenolsulphonphthalein in the Cerebrospinal System, J. A. M. A. **79**:1014 (Sept. 23) 1922. Dandy, W. E.: Treatment of Staphylococcus and Streptococcus Meningitis by Continuous Drainage of Cisterna Magna, Surg., Gynec. & Obst. **39**:760 (Dec.) 1924. Hughson, W.: Embryogenesis of Human Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **14**:240 (Aug.) 1925. Fremont-Smith, F., and Hodgson, J. S.: Combined Ventricular and Lumbar Puncture, in Dana, C. L.; Davis, T. K., and others: Human Cerebrospinal Fluid: An Investigation of the Most Recent Advances as Reported by the Association for Research in Nervous and Mental Diseases (series of research publications, 1924, vol. 4, p. 122), New York, Paul B. Hoeber, Inc., 1926. Masserman and Schaller.[1]

17. Discounting the possible minor local variations due to active secretion or absorption of the cerebrospinal fluid.

18. This correspondence of ventricular, cisternal and lumbar pressures has been demonstrated by: (*a*) Claude, H.: Bull. et mém. Soc. méd. d. hôp. de Paris **28**: 319, 1912. (*b*) Dahlstrom, S., and Wideroe, S.: Ztschr. f. d. ges. Neurol. u. Psychiat. **72**:75, 1921. (*c*) Weigeldt, R. W.: Studien zur Physiologie und Pathologie des Liquors cerebrospinalis, Jena, Gustav Fischer, 1923. (*d*) Ruggles, A. H.: Observations in Ventricle and Cistern Punctures, Arch. Neurol. & Psychiat. **11**: 227 (Feb.) 1924. (*e*) Ayer, J. B., and Solomon, H. C.: Cerebrospinal Fluid from Different Loci, ibid. **14**:303 (Sept.) 1925. (*f*) Cushing, H.: Studies in Intracranial Physiology and Surgery, New York, Oxford University Press, 1925.

subarachnoid system would vary directly as the pressure recorded by a manometer connected to the lumbar subarachnoid space; moreover, any sudden decrease in pressure at this locus would almost immediately be reflected in all other portions of the system. The readings of pressure in the lumbar subarachnoid space in patients in the horizontal position may therefore be considered to equal pressures that obtain at the time throughout the other ventriculomeningeal spaces provided the amount of fluid withdrawn has not been so large as to cause actual collapse of any part of the subarachnoid system.

BASIC CEREBROSPINAL FLUID PRESSURE

It is not one of the purposes of the present article to discuss the interrelationships of the basic pressure of the cerebrospinal fluid with other variables such as age, clinical condition, dP, Tr, etc.; the correlations that may be deduced from the data will be made the subject of a later report. However, as has been pointed out in table 1, a simple comparison of means reveals a striking uniformity of basic cerebrospinal fluid pressure for the contrasted colored, white, male, female, control patients and patients with dementia paralytica. The grand mean of the entire series (147.71 ± 1.89 mm.) corresponds well with the average spinal pressure in the horizontal position reported by most other investigators,[19] although it is higher than that considered normal by Fay.[4] The mean pressure in the sitting position (397.6 ± 4.2 mm.), on the other hand, is somewhat less than that reported by Howe[20] and greater than the criterion of "double the lumbar pressure" of Ayer.[21] These differences, however, may be accounted for by the marked influence on the cerebrospinal fluid pressure occasioned by the great variation in posture possible in the "sitting position"—a circumstance that renders the comparison of various series inaccurate unless the "sitting posture" of patients in the several series was kept fairly constant.

PHYSICAL CONCEPTS OF INTRACRANIAL HYDRODYNAMICS

Any further discussion of the hydrodynamics of the central nervous system must necessarily involve the consideration of a hypothesis that

19. (*a*) Bungart: Die Messung des Hirn- bzw. Lumbaldruckes mittels Quecksilbermanometer, Psychiat.- neurol. Wchnschr. **18**:391, 1916-1917. (*b*) Weigeldt,[18c] p. 33. (*c*) Ayer, J. B., in Dana, C. L.; Davis, T. K., and others: Human Cerebrospinal Fluid: An Investigation of the Most Recent Advances as Reported by the Association for Research in Nervous and Mental Diseases (series of research publications, 1924, vol. 4, p. 159), New York, Paul B. Hoeber, Inc., 1926.

20. Howe, H. S.: Physiologic Mechanism for Maintenance of Intracranial Pressure, Arch. Neurol & Psychiat. **20**:1048 (Nov.) 1928.

21. Ayer, J. B.: Cerebrospinal Fluid Pressure from the Clinical Point of View, Arch. Neurol. & Psychiat. **14**:440 (Oct.) 1925.

has long dominated thought in this field—the so-called Monro-Kellie doctrine. In 1783, Monro,[22] on purely anatomic grounds, first advanced the postulate that "as the brain is enclosed in a case of bone" the volume of which is constant, and that "as the substance of the brain like that of other solids in the body is nearly incompressible, the quantity of blood within the head in different positions must be the same, or very nearly the same." Kellie,[23] about forty years later, presented certain experimental support of this reasoning, and the thesis soon found acceptance by other investigators.[24]

Neither Monro nor Kellie, however, had taken the possible hydrodynamics of the cerebrospinal fluid into account, although this medium had been described by Cotugno[25] as early as 1764. Burrows[26] was the first to correct this deficiency partially by postulating that as a matter of fact the intracranial volume of blood was not constant, but varied inversely as the volume of the cerebrospinal fluid. It soon became evident, moreover, that since the spinal subarachnoid spaces communicated freely with those of the cranium,[15a] the hydrodynamics of the two divisions of the craniovertebral cavity were intimately related. The original Monro-Kellie hypothesis was therefore modified to state that, since the dura was completely enclosed by the rigid craniovertebral canal, the gross volume of intradural contents remained unchangeable; and further, since the tissue of the central nervous system itself is incompressible,[27] the volumes of blood and cerebrospinal fluid within the dural sac must vary in inverse relationship to each other.

In this form the "doctrine" received what appeared to be considerable experimental confirmation. Thus, Weed and McKibben[28] dem-

22. Monro, A.: Observations on the Structure and Functions of the Nervous System, Edinburgh, Creech and Johnson, 1783.

23. Kellie, G.: Tr. Med.-Chir. Soc., Edinburgh **1**:84, 1824.

24. (*a*) Abercrombie, John: Pathological and Practical Researches on Diseases of the Brain and the Spinal Cord, Edinburgh, Waugh & Innes, 1828. (*b*) Ecker, A.: Physiologische Untersuchung über die Bewegungen des Gehirns und Rückenmarks, Stuttgart, E. Schweizerbart, 1843. (*c*) Hill, L.: Physiology and Pathology of the Cerebral Circulation, London, J. & A. Churchill, 1896.

25. Cotugno, quoted by Craig, C. B.: The Normal Cerebrospinal Fluid: A Review of the Literature, in Dana, C. L.; Davis, T. K., and others: Human Cerebrospinal Fluid: An Investigation of the Most Recent Advances as Reported by the Association for Research in Nervous and Mental Diseases (series of research publications, 1924, vol. 4, p. 22), New York, Paul B. Hoeber, Inc., 1926.

26. Burrows, G.: On Disorders of the Cerebral Circulation, London, Longman [and others], 1846.

27. Becht: Am. J. Physiol. **51**:1, 1920. Howe.[20]

28. Weed, L. H., and McKibben, P. S.: Am. J. Physiol. **48**:512, 1919; ibid. **48**:531, 1919; Anat. Rec. **16**:167, 1919.

onstrated that whereas the intravenous injection of strongly hypertonic salt solution into an intact animal produced marked shrinkage of the brain and negative pressures in the spinal fluid, pressures lower than atmospheric could not be obtained if the protection afforded by the bony craniovertebral box was removed by opening the calvarium.[29] Again, Kubie and Hetler [30] showed that the capillary bed of the cerebral surface became engorged in compensation for the diminution in cerebrospinal fluid volume produced by the injection of hypertonic solutions intravenously. This was confirmed for the intracranial vascular system in general by Weil, Zeiss and Cleveland,[31] who employed chemical analysis of the cranial blood stream and concluded that the intravenous injection of hypertonic solutions produced an increase in the volume of intracranial blood. Such research, however, dealt only with gross changes in the relative volumes of the intradural contents, and did not determine whether small diminutions in the total volume of the dural sac could or could not occur in the presence of an uninjured craniovertebral canal. The possibility remained, therefore, that even in an intact animal the exactly inverse relationships of the volume of the blood and the cerebrospinal fluid postulated by the modified Monro-Kellie doctrine could be prevented by slight changes in the total volume of the dural envelop, since variations in this volume were theoretically possible through the operation of mechanisms such as the movement of spinal fluid along nerve sheaths, the elasticity of the occipito-atlantal ligament and readjustments in the volume of blood of extradural vessels, especially the spinal venous plexuses. Dixon and Halliburton,[32] employing the experimental methods of Hill,[24c] had in fact concluded that "the cranial contents cannot any longer be regarded as a fixed quantity without the power of expanding or contracting in volume," but this conclusion did not appear wholly justified by their evidence.

Weed and his associates attacked this problem in a succession of ingenious investigations. In their initial experiments [33] they observed that

29. Weed, L. H., and Hughson, W.: The Cerebrospinal Fluid in Relation to the Bony Encasement of the Central Nervous System as a Rigid Container, Am. J. Physiol. **58**:85, 1921; ibid. **58**:101, 1921.

30. Kubie, L. S., and Hetler, D. M.: The Cerebral Circulation; IV. The Action of Hypertonic Solutions, Arch. Neurol. & Psychiat. **20**:749 (Oct.) 1928.

31. Weil, A.; Zeiss, F. R., and Cleveland, D. A.: Am. J. Physiol. **98**:363, 1931.

32. Dixon, W. E., and Halliburton, W. D.: J. Physiol. **48**:128, 1914.

33. Weed, L. H., in Elsberg, C. A., and others: Intracranial Pressure in Health and Disease: An Investigation into the Most Recent Advances as Reported by the Association for Research in Nervous and Mental Diseases (series of research publications, vol. 8, p. 25; 1927, vol. 8, p. 25), Baltimore, Williams & Wilkins Company, 1929. Some Limitations of the Monro-Kellie Hypothesis, Arch. Surg. **18**:1049 (April) 1929. Weed, L. H., and Flexner, L. B.: Further Observations on the Monro-Kellie Hypothesis, Bull. Johns Hopkins Hosp. **50**:196, 1932.

in a series of dogs whose cranial dura had been widely exposed by trepanation a head-down tilt caused no greater change in the cisternal fluid pressure than did a corresponding tilt in intact animals; tilting the dogs in a tail-down position, however, caused a greater change of cisternal pressure in the intact animals. Conversely, in a series with the vertebral canal opened, the changes in pressure in the cisternal fluid on tilting from the horizontal to the vertical tail-down position were of about the same magnitude as those that occurred in a series of animals that had not been operated on, whereas on vertical head-down tilts the changes in pressure in the series operated on were considerably greater than those that occurred in the intact animals. From these data the inference appeared obvious that whereas both the cranial and the spinal dura are practically indistensible and therefore relatively independent of outside bony support, the presence of an intact craniovertebral covering is necessary to prevent the collapse of either portion of the dura when the pressure within it becomes less than the atmospheric pressure without.

Weed and his co-workers then developed an experimental method for studying further the effects of changes in intradural volume on pressure. They first discovered [34] that the alterations of pressure in a dog's basal cistern produced by tilting the animal were less, as measured in open-end manometers through which fluid could be displaced into or out of the dog's subarachnoid system during the tilts, than were the corresponding changes in pressure in "bubble-manometers" which measured cisternal pressure without the displacement of fluid. They then observed that if the amount of fluid (dV) displaced into or out of an individual open-end manometer during a single tilt was divided by the difference in pressure (dP) between the reading of that manometer and the reading of a bubble manometer for a comparable tilt, then "calculating this fraction dV/dP from the results of the tiltings to the vertical head-down and tail-down positions, a fairly constant value (average = 0.17 in dogs weighing from 6 to 7 Kg.) was obtained in most experiments." These findings led the authors to compare the coverings of the central nervous system with an elastic container in which changes in pressure were, within the limits of its elasticity, directly proportional to changes in volume. Employing the physical formula for the coefficient of elasticity (E = stress/strain) and substituting the values of dP for stress and dV/V for strain, Weed and his co-workers obtained the biometric equation $E = \frac{dP}{dV} V$, in which E represented "the coefficient of volume elasticity of the dural envelop," dP and dV the values obtained on manometric readings in tilting experiments and V "the volume of the intradural contents." Employing in this formula the experimental value dV/dP = 0.17, they were then able to predict, from the measurement of the decrease in cerebrospinal fluid pressure produced by the intravenous injections of hypertonic solutions, the change in "intradural" volume concurrently produced by these injections. In further experiments they [35] determined a value for V in their formula

34. Weed, L. H.; Flexner, L. B., and Clark, J. H.: The Effect of Dislocation of Cerebrospinal Fluid on Its Pressure, Am. J. Physiol. **100**:246, 1932.

35. Flexner, L. B.; Clark, J. H., and Weed, L. H.: The Elasticity of the Dural Sac and Its Contents, Am. J. Physiol. **101**:292, 1932.

by combining the volume of mercury held by the empty cranium with the volume of oil necessary to fill the empty vertebral dural sac of their experimental animal, and thus calculated the average value of E in their experimental animals as ranging from 3.8×10^5 dynes per cubic centimeter for old dogs to 4.6×10^5 dynes per cubic centimeter for young ones. Weed and his associates then demonstrated that any one factor precalculated by their formula could be checked by experiment, and further showed [36] that the same formula could be applied to the dural envelop of the cat and of an essentially erect primate—the macaque—in which animals E showed an age variation corresponding to that demonstrated in dogs. Summarizing and interpreting this work with regard to the original Monro-Kellie hypothesis, Weed [37] recently restated this doctrine as follows: "With a known elastic element now introduced into the concept, it is possible to speak with accuracy of the fairly constant content of the cranial and spinal dural sacs—which is after all almost what Alexander Monro said one hundred and fifty years ago."

AYALA'S QUOTIENTS

Even before the publication of this research Ayala [38] had observed clinically that the removal of a given quantity of spinal fluid reduced its pressure to a greater degree in patients in whom the volume of the ventriculosubarachnoid spaces was encroached on (as in tumor or edema of the brain) than did the removal of the same amount of fluid in cases in which the system was dilated (hydrocephalus or atrophy of the brain). He then advanced the theorem that if the product of the final cerebrospinal fluid pressure and the quantity of fluid removed be divided by the initial cerebrospinal fluid pressure ($F \times q/I$) the resulting *Rachidealquotient* (Rq) would indicate numerically the relative volume of the ventriculosubarachnoid spaces at the time of drainage. Ayala calculated this quotient in a series of patients with various diseases of the nervous system, and showed that, in general, low values (0.62) were obtained in cases of spinal subarachnoid block, higher values (2.25 to 4.25) in cases of tumor of the brain and maximum values (10 or higher) in cases of hydrocephalus. In a later paper, however, Ayala [39] took into account the fact that the extent of the fall in the pressure of the cerebrospinal fluid following drainage is related not alone to the volume of fluid removed, but also to the patency of the ventriculosubarachnoid system, the volume of blood entering and leaving the cranium and the elasticity of the meninges. Ayala then revised his formula to read $Q = I - F/v$ (initial cerebrospinal fluid pressure minus final cerebrospinal fluid pressure divided by the volume of spinal fluid removed at drainage)—the Q of this formula standing in inverse relationship to his original Rq and measuring in combination the relative decrease in volume of the ventriculosubarachnoid spaces and the coefficient of meningeal elasticity. Since, however, Ayala considered the latter function to vary only within relatively narrow limits, he placed considerable reliance on the ability of his second formula to indicate the

36. Weed, L. H., and Flexner, L. B.: Cerebrospinal Elasticity in the Cat and Macaque, Am. J. Physiol. **101**:668, 1932.

37. Weed, L. H.: Positional Adjustments of the Pressure of the Cerebrospinal Fluid, Physiol. Rev. **13**:80, 1933; Some Aspects and Problems of Intracranial Pressures, Bull. Johns Hopkins Hosp. **52**:345 (May) 1933.

38. Ayala, G.: Ueber den diagnostischen Wert des Liquordruckes und einen Apparat zur seiner Messung, Ztschr. f. d. ges. Neurol. u. Psychiat. **84**:42, 1923.

39. Ayala, G.: Monatschr. f. Psychiat. u. Neurol. **58**:65, 1925.

relative volume of the ventriculosubarachnoid system in his patients, and showed that its Q varied from a value of 1.5 in cases of serous meningitis to one of 4.5 in cases of tumor of the brain.

INTERPRETATION OF FORMULAS

The data of the present study confirm Ayala's observations only in part. While, in general, abnormally large decreases in cerebrospinal fluid pressure per volume of fluid removed were noted in cases of acute alcoholic cerebral edema and in 4 cases of tumor of the brain later confirmed at autopsy,[8] it must be noted that the pressure index dV/dP (the inverse of Ayala's Q) was not constant even in the control series, but varied within fairly wide limits from patient to patient. Further, the mean value of dV/dP was somewhat less for drainages of 10 cc. than for drainages of larger amounts (table 2), and appeared to be relatively fixed only for drainages of from 20 to 30 cc. of fluid, between which limits the cerebrospinal vascular readjustments compensatory to the drainages of fluid in the various series seemed most constant. Again, the mean dP_1 produced by the drainage of 35 cc. of spinal fluid in a series of 27 patients with advanced dementia paralytica (in whom cerebral atrophy and consequent increase of the volume of subarachnoid fluid appeared probable) was 24.9 ± 5.35 mm. greater than the corresponding dP_1 in a series of 32 controls, indicating that in cases of dementia paralytica at least the increase in meningeal elasticity overbalances the effect of the dilatation of the ventriculomeningeal spaces and gives misleadingly low values to the pressure index of the series and high values to Ayala's Q. It must be inferred, therefore, that although Ayala's formulas may be clinically useful in indicating large pathologic deviations from the average in the volume of the ventriculomeningeal system, they cannot be applied with accuracy to determine the relative sizes of this system in a series of normal persons. Moreover, the formulas may also be misleading when applied to patients with complex pathologic alterations in the brain and meninges, probably because of the variations in the coefficient of volume elasticity of the ventriculomeningeal system that occurs in these cases concurrently with the change in volume of the ventriculosubarachnoid spaces.

These considerations, however, need not obscure certain observations that indicate that both the pressure index and the coefficient of elasticity of meningeal volume remain fairly constant in an individual person under comparatively wide variations of intracranial hydrostatics. Weed's work on experimental animals, of course, points in this direction, but a firmer basis than the a priori application of Weed's formula to man is available in certain of the data of the present series. Thus, in a series of experiments[8] in which patients were tilted, and the value dV/dP for relatively small displacements of spinal fluid determined according to a modification of the method of Weed and his co-workers, the pressure index for any one patient was found to vary only within comparatively narrow limits for each positional tilt. As previously noted, also, a similar constancy of the pressure index was observed in a complementary series of experiments in which successive amounts of 5 or 10 cc. of spinal fluid were removed from a single patient. Moreover, the results of the experiments in which drainages in the same patient were repeated at daily or weekly intervals indicate that the pressure index for any patient remains fairly stable over relatively long periods. With V constant and the drainage index thus demonstrated to be comparatively stable, the elasticity of meningeal volume in each patient may justifiably be considered also to have remained relatively fixed. Consequently, the formula for the coefficient of elasticity of volume, $E = \frac{dP}{dV} V$,

shown by Weed to hold for the individual dog and macaque, appears to be also applicable to the individual person.

Before applying this formula to the data of this series, however, it seems necessary to clarify the significance of two of its quantities, viz., E and V. In determining the value of V for his animals, Weed and his associates measured the total volume of the craniovertebral dural envelop. When it is considered, however, that both his experiments and those reported here were concerned with the effects of displacement of cerebrospinal fluid on its pressure, and that the elastic vessel which holds this fluid is not the dural envelop as a whole but the much more complex system of ventriculosubarachnoid-perivascular spaces and channels, it becomes apparent that the value of V may, for present purposes, well be revised. Hydrodynamically the ventriculosubarachnoid system is bounded only at its periphery by the dura—a fibrous envelop which possesses practically no distensibility and a normal collapsibility determined in great part by the corresponding inward compressibility of the internal walls of the ventriculosubarachnoid spaces. The boundaries of these spaces, on the other hand, are both highly elastic and highly complex, including as they do the ramifications of the ventricles, the extensions along nerve sheaths and the arborizations of the Virchow-Robin channels about the blood vessels of the brain and spinal cord. It is, in fact, through the direct contact of the layers of the arachnoid with the elastic walls of the blood vessels of the neuraxis that the ventriculosubarachnoid system as a whole derives the greater portion of its specific elasticity of volume—a property which the peripheral dural envelop would scarcely possess of itself. The effects of the displacement of spinal fluid on its pressure within this system, therefore, are logically much more closely related to the specific volume of the ventriculosubarachnoid spaces (V_{vs}) than to the total volume (V) of the dural envelop eviscerated of the brain, blood vessels, nerves, spinal fluid, etc. This concept becomes all the more evident in its clinical application when it is considered that if Ayala's Q and Rq—which are functions of E—were not related to ventriculosubarachnoid rather than to dural volume the calculation of his quotients would have little clinical significance, as total intradural volume can necessarily vary but little even in extensive disease of the central nervous system. Similarly, since dV in the data of this series measures the diminution in volume and dP the resultant diminution in pressure in the ventriculomeningeal spaces after the drainage of cerebrospinal fluid, V must be made to equal the volume (V_{vs}) and E the specific coefficient of elasticity (E_{vs}) of the unitary ventriculosubarachnoid system rather than of its outer dural mantle.[40] Weed's formula may then be modified for the purpose to read: $E_{vs} = V_{vs}\frac{dP_1}{dV}$, or the coefficient of elasticity of a patient's ventriculosubarachnoid system equals the product of

40. If V and E are made to equal, respectively, the volume and the coefficient of volume elasticity of the ventriculosubarachnoid system, certain of the inconsistent results in the experiments reported by Weed, Flexner and Clark may be accounted for. Thus, in an occasional experiment (four or twenty in one series) these authors found that the value of dV/dP varied considerably between head-down and tail-down tilts in the same animal, and the fraction was also found to vary for different levels of anesthesia. Weed explained these deviations as being due to " a difference in elasticity [in the dural envelop] for the two vertical positions." It is difficult to conceive of such changes of elasticity occurring in the dural envelop, but they could easily occur in the ventriculosubarachnoid system in response to variations in the distribution of the volume of vascular blood in the different positions.

the volume of that system and the inverse of the patient's pressure index as determined in the observations in his case. Further, since cerebrospinal vascular engorgements produced by the successive fractional drainage of as much as 50 cc. of spinal fluid from an individual patient cause, as already noted, no significant change in the value of dV/dP, then E_{vs} is also constant for successive drainages of spinal fluid in the same position of the patient, and $E_{vs} = \frac{dP_2}{dV} V_{vs2}$ when V_{vs2} represents the volume of the ventriculosubarachnoid system at the time of the second drainage of dV, and dP_2 the fall in cerebrospinal fluid pressure produced by this drainage. Since dV was held constant in the experiments these formulas may be combined to obtain $V_{vs2}/V_{vs} = dP_1/dP_2$, from which it may be seen that the ratio of the volume of the ventriculomeningeal spaces at the time of the first drainage of cerebrospinal fluid to the volume of these spaces at the second drainage of the same amount of fluid stands in inverse relationship to the extent of the diminution in cerebrospinal fluid pressure respectively effected by the two drainages. Or, more specifically interpreted, if the fall in pressure of the cerebrospinal fluid is significantly greater during the second drainage of spinal fluid than during the first, then the volume of the ventriculosubarachnoid spaces at the time of the second drainage is less than it was at the time of the first removal of fluid.

APPLICATION OF FORMULAS

The formulas in the preceding section have been derived from those of Weed and Ayala in order to render mathematically demonstrable an important clinical conclusion from the data of this series, namely, that, since dP_2 for dV =35 cc. is significantly (23.5 ± 4.5 mm.) greater than the corresponding dP_1 (table 2), the rapid drainage of 35 cc. of cerebrospinal fluid produces a constriction of the ventriculosubarachnoid spaces which is still persistent from one to three hours after the drainage of fluid and probably for a number of hours thereafter. The significance of this conclusion becomes more apparent when it is considered that, even according to this modified conception of the Monro-Kellie doctrine, there can be but two clinically relevant causes and (or) accompaniments of this marked diminution in ventriculosubarachnoid volume, first, persistent craniovertebral vascular dilatation, and (or) second, swelling of the tissue of the central nervous system. That persistent vascular engorgement is produced by the rapid drainage of spinal fluid has already been indicated in one series of experiments by the cumulative increases in cerebrospinal fluid pressure that result when the fluid is drained and reinjected. Proof of the concurrent production of edema of the central nervous system is not directly deducible from the present data, although the occurrence of edema in the tissue could readily explain the transient symptoms and signs of disturbed physiology in the central nervous system that are produced by the rapid drainage of cerebrospinal fluid. However, more direct evidence on this point was furnished in a series of experiments [11] in which hypertonic solutions of dextrose were administered intravenously to each patient fol-

lowing the rapid removal of large amounts of spinal fluid; in contrast to the control findings previously discussed dP_2 for $dV = 35$ cc. in the dextrose series was not significantly greater than dP_1. Since the administration of hypertonic solutions of dextrose intravenously either does not directly affect [41] or increases [42] the volume of the intracranial vessels, but undoubtedly decreases the volume of the nervous tissue,[43] these experiments indicate that in the series of spinal drainages in which dextrose was withheld both engorgement of the vessels and edema of the central nervous system were produced by the rapid drainage of large amounts of cerebrospinal fluid. This confirms for man a similar finding previously deduced from experiments on animals [2] and furnishes a conclusion of obvious clinical importance.

INTERPRETATION OF Tr INTERVALS

If the amount of spinal fluid (dV) drained at any puncture is divided by the time required (Tr) for the restoration of the pressure of the fluid to its level before the drainage, the quotient dV/Tr will express the mean rate of replacement of spinal fluid by secretion in cubic centimeters per minute, provided all other factors in intracranial hydrostatics remained or were restored to normal during the Tr interval. As has been indicated, this provision appears to hold true for drainages of 20 cc. or less of fluid, so that the quotient for this dV (0.305 cc. per minute, table 3) probably most closely approximates the true rate of replacement of cerebrospinal fluid after the removal of comparatively small amounts. Quotients for drainages of larger amounts are higher (table 3), and consistently so, since under these circumstances there is probably not only a greater stimulus to the secretion of spinal fluid, but also a diminution in the volume of the ventriculosubarachnoid

41. Howe, H. S.: Microcinematographic Studies of the Effect of Certain Drugs on the Cerebral Circulation, Arch. Neurol. & Psychiat. **28**:944 (Oct.) 1932.

42. Kubie and Hetler.[30] Weil, Zeiss and Cleveland.[31]

43. Weed, L. H., and McKibben, G. S.: Am. J. Physiol. **48**:512, 531, 1919; Anat. Rec. **16**:167, 1919. Haden, R. L.: Therapeutic Application of the Alteration of Brain Volume, J. A. M. A. **78**:983 (Sept. 27) 1919. Foley, F. E. B., and Putnam, T. J.: Am. J. Physiol. **53**:464, 1920. Ebaugh, F. G., and Stevenson, C. S.: Bull. Johns Hopkins Hosp. **31**:440, 1920. Sachs, E., and Malone, J. Y.: Am. J. Physiol. **55**:277, 1921. Dowman, C. E.: Management of Head Injuries in the Real or Potential Brain Damage, J. A. M. A. **79**:2212 (Dec. 30) 1922. Howe, H. S., in Dana, C. L.; Davis, T. K., and others: Human Cerebrospinal Fluid. An Investigation of the Most Recent Advances as Reported by the Association for Research in Nervous and Mental Diseases (series of research publications, 1924, vol. 4, p. 207), New York, Paul B. Hoeber, Inc., 1926. Masserman, J. H.: Effects of Intravenous Administration of Hypertonic Solutions of Dextrose, J. A. M. A. **102**:208 (June 23) 1934.

spaces which it is necessary to distend before the pressure of the cerebrospinal fluid is restored to its original level.

It must, of course, be remembered that the values calculated from the data for the rate of replacement of cerebrospinal fluid after drainage may have no correlation with the rate of dialysis and/or secretion of the fluid in the absence of this stimulus. The "normal" rate of production of cerebrospinal fluid is not definitely known for man;[44] estimates vary from 45 to 65 cc. per day (0.031 to 0.045 cc. per minute) in patients on a daily water intake[45] of 900 cc. to from 0.5 to 1 liter per day (0.346 to 0.692 cc. per minute) in patients with unregulated water intake.[46] That the rate may also vary under different physiologic conditions is indicated by the large deviations in the quotient dV/Tr as determined in experiments on the same patient from week to week.

One other conclusion is afforded by the data: If, in table 3, the weighted average of dV/Tr_2 for drainages of from 20 to 35 cc. of fluid (0.376 cc. per minute) is compared with the weighted average of dV/Tr_1 for the same amounts (0.319 cc. per minute), the former is found to exceed the latter by about 18 per cent, indicating the stimulative effect of the second drainage of cerebrospinal fluid on the rate with which it is replaced. A similar conclusion can be deduced from the fact that the mean Tr_2 is significantly greater than the mean Tr_1 (table 3), for $dV = 35$ cc.—the only two individual Tr series large enough to justify this comparative analysis.

RELATIONSHIP OF CEREBROSPINAL FLUID PRESSURE TO ARTERIAL BLOOD PRESSURE

There is considerable disagreement in the literature with respect to the relationship of the systemic arterial blood pressure to the pressure of the cerebrospinal fluid. Fremont-Smith and Merritt[47] in a study of simultaneous blood pressure and cerebrospinal fluid pressure readings in 1,600 patients, concluded that "The level of cerebrospinal fluid pres-

44. Fremont-Smith, F.: Pathogenesis of the Changes in the Cerebrospinal Fluid in Meningitis, Arch. Neurol. & Psychiat. **28**:779 (Oct.) 1932. Estimates based on the fact that it takes about seventy-two hours for the ventriculosubarachnoid system to refill after encephalography (Grant, F. C.: Ventriculography and Encephalography, Arch. Neurol. & Psychiat. **27**:1310 [June] 1932; Fay[4]) are obviously inaccurate, since the evacuation of the fluid and the presence of the unabsorbed air may well influence the rate of reformation of the cerebrospinal fluid.

45. Fay, T., in discussion of Cobb.[46]

46. Cobb, S.: Causes of Epilepsy, Arch. Neurol. & Psychiat. **27**:1245 (May) 1932; correction, ibid. **28**:172 (July) 1932.

47. Fremont-Smith, F., and Merritt, H. H.: The Relationship of Cerebrospinal Fluid Pressure to Systemic Blood Pressure, abstr., J. Nerv. & Ment. Dis. **78**:291 (Sept.) 1933.

sure has no appreciable effect on systemic blood pressure unless the cerebrospinal fluid pressure is greatly increased so as to approach the level of the diastolic blood pressure." Wilson,[48] in a comparable investigation of 100 cases of tumor of the brain, reached similar conclusions. It must be considered, however, that studies of simultaneous readings of blood and cerebrospinal fluid pressure in large series of cases taken in static cross-section give no exact information as to the possible dynamic interrelationships of the two pressures over short periods of time in an individual patient. As a matter of fact, there is considerable experimental and clinical evidence[49] that the respective deviations in the pressure of the blood and cerebrospinal fluid in response to transient physiologic changes are to a certain extent mutually interdependent.

Unfortunately, the data of this series on this point cannot be too closely interpreted. As was noted in one of the series (table 4, series 7), the mean systolic blood pressure dropped about 9 mm. of mercury following the marked decrease in spinal fluid pressure (126.6 ± 4.7 mm.) occasioned by the removal of 20 cc. of spinal fluid in the sitting position. In another series (table 4, series 5) both the systolic and the diastolic blood pressures rose, respectively, 7 and 9 mm. above normal during the period of intracranial hypertension which, as has been shown, begins approximately three hours after the rapid drainage of from 35 to 50 cc. of spinal fluid. While these relationships seem to indicate a measure of direct correspondence between the pressures of blood and cerebrospinal fluid, it must nevertheless be considered that the rapid reduction of cerebrospinal fluid pressure of itself causes definite physiologic effects in the central nervous system, and could in this manner influence the blood pressure entirely apart from the direct effect of the drainage on cerebrospinal fluid tension. The correspondence of the blood and cerebrospinal fluid pressures in the data may therefore be fortuitous and not entirely attributable to their mutual physiologic relationship.

POSTPUNCTURE SYMPTOMATOLOGY

The relationship of postpuncture symptoms to the occurrence of vascular congestion and edema of the central nervous system has been discussed in a previous paper,[8] and need not be extensively recapitulated here. It may be well, however, to point out certain relationships between

48. Wilson, A. A.: Blood Pressure Observations in Tumors of the Brain, Arch. Neurol. & Psychiat. **28**:891 (Oct.) 1932.

49. Cushing, H.: Am J. M. Sc. **124**:376, 1902. Bailliart; Magniel, and Saragea: Arch. d. mal. du cœur **17**:289, 1924. Block, E. B., and Oppenheimer, R. H.: A Comparative Study of Intraspinal Pressure, Blood Pressure and Intraocular Tension, Arch. Neurol. & Psychiat. **11**:444 (April) 1924. Ayer.[19c] Howe.[20] Marx, E.: The Simultaneous Registration of the Intraocular Pressure and the Pressure of the Cerebrospinal Fluid, Ann. d'ocul. **168**:125 (Feb.) 1931.

the rapid drainage of cerebrospinal fluid and the occurrence of the postpuncture syndrome that may be deduced with especial significance from the experiments in the present series.

Primarily it is evident that severe disturbances of the fluid balance of the central nervous system are not requisite to the production of untoward postpuncture effects in every case, since headaches and other mild symptoms occasionally occurred in patients from whom as little as 10 cc. of fluid was drained. In these cases, however, the headaches nearly always followed immediately after the puncture, and were frequently benefited by lowering the patient's head, so that their etiologic basis may well have been a transient intracranial hypotension. On the other hand, the incidence and severity of postpuncture symptoms increased in direct ratio to the extent and rapidity of the reduction in cerebrospinal fluid pressure at the time of drainage;[50] further, adverse symptoms beginning two hours or more after the puncture could not be attributed to intracranial hypotension, since the cerebrospinal fluid pressure following the removal of even large amounts of fluid returned to its original level within one or two hours, and then mounted above. Finally, postpuncture symptoms were almost inevitably produced in the experiments in which the occurrence of subarachnoid hypotension was prevented by the immediate reinjection of the fluid after drainage. It therefore becomes highly probable that the deferred untoward symptoms caused by the rapid removal of large amounts of spinal fluid are due in the majority of cases not alone to a simple mechanical alteration in subarachnoid hydrostatics but also to the congestion and edema of the central nervous system that result from severe disturbances in its fluid balance.

SUMMARY AND CONCLUSIONS

A method is presented for studying certain phases of cerebrospinal hydrodynamics in man, and the results of various series of experiments are analyzed as far as possible according to a standard statistical technic. Full cognizance is taken of the fallibility of even "statistically significant" constants derived from experiments with such highly variable samplings as small series of human beings.[51] Nevertheless, within the limits of experimental and statistical error the following conclusions appear justified:

1. The pressure of the cerebrospinal fluid under basic conditions of body relaxation either remained nearly constant during the observation periods of from two to six hours or showed very slow variations in response to undetermined physiologic changes.

50. This observation has also been made by B. J. Alpers (Lumbar Puncture Headaches, Arch. Neurol. & Psychiat. **14**:806 [Dec.] 1025).

51. Wilson, E. B.: The Statistical Significance of Experimental Data, Science **58**:93, 1923.

2. There were no significant differences in basic cerebrospinal fluid pressure among the several series of female, male, white, colored and control patients and patients with dementia paralytica.

3. The basic cerebrospinal fluid pressure in an individual patient may vary considerably over long intervals in response to changing physiologic conditions.

4. Following the drainage of from 10 to 50 cc. of spinal fluid its pressure returns to normal in a mean time of from one to three hours. If more than 30 cc. of spinal fluid is rapidly removed its pressure following restoration to normal mounts to a higher level for from eight to twenty-four hours.

5. After the drainage of 20 cc. of cerebrospinal fluid the latter is replaced at the rate of about 0.3 cc. per minute. The drainage of spinal fluid in amounts larger than 20 cc. probably causes a slightly more rapid rate of replacement. The rate is also accelerated by the second successive drainage of cerebrospinal fluid, but not by subsequent drainages.

6. The rapid removal of 10 cc. or more of spinal fluid causes a vascular dilatation in the central nervous system which does not completely regress if the spinal fluid is immediately reinjected.

7. The rapid drainage of 35 cc. or more of spinal fluid causes a constriction in the size of the ventriculosubarachnoid spaces which begins immediately after the drainage and persists for eight hours or longer.

8. This diminution in the size of the ventriculomeningeal spaces is attributable to a persistent vascular engorgement of the central nervous system and probably also to edema of tissue of the central nervous system.

9. The rapid drainage of 35 cc. or more of cerebrospinal fluid may cause, in addition, symptoms and signs of meningeal irritation and sterile pleocytosis of the spinal fluid.

10. The severity of the late postpuncture syndrome, characterized variously by headache, backache, vertigo, nausea, fever and signs and symptoms of meningeal irritation, is directly related to the intensity of the postpuncture physiopathologic changes in the central nervous system.

PHYTOTOXIC INDEX

I. RESULTS OF STUDIES WITH SIXTY-EIGHT MALE SCHIZOPHRENIC PATIENTS

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Many eminent students of the schizophrenic psychosis have postulated for it a toxic genesis. Many statements have appeared in the literature that have been interpreted as tending to substantiate this theory, while an equally formidable number has been presented tending to refute it. Because of these conflicting opinions, tests for this hypothetic toxin were undertaken in conjunction with the intensive study of schizophrenia as outlined by Hoskins and his co-workers.[1]

The use of viable plant protoplasm as a toxicity test was first recorded by Kahlenberg and True,[2] but it was not until twenty-six years later that this method of study (phytopharmacology) became popular in this country.[3] Macht [4] reported that the blood of patients with pernicious anemia had a decided inhibitory action on the growth of seedlings of Lupinus albus, so much so that he recommended this test as a diagnostic point differentiating between pernicious anemia and other blood dyscrasias.

The application of this method to psychotic patients was first proposed by Looney [5] who, in conjunction with Macht, determined the

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From the Worcester State Hospital and memorial foundation for neuroendocrine research. With the collaboration of the research staff of the Worcester State Hospital.

1. Hoskins, R. G.; Sleeper, F. H.; Shakow, D.; Jellinek, E. M.; Looney, J. M., and Erickson, M. H.: A Cooperative Research in Schizophrenia, Arch. Neurol. & Psychiat. **30**:388 (Aug.) 1933.

2. Kahlenberg, L., and True, R. H.: On the Toxic Action of Dissolved Salts and Their Electrocytic Dissociation, J. A. M. A. **27**:138 (July 18) 1896.

3. Macht, D. I., and Livingston, M. B.: Effect of Cocaine on the Growth of Lupinus Albus, J. Gen. Physiol. **6**:573, 1922.

4. Macht, D. I.: Phytotoxic Properties of Blood in Pernicious Anemia, J. Pharmacol. & Exper. Therap. **29**:461, 1926.

5. Looney, J. M., and Macht, D. I.: Relation Between the Undetermined Nitrogen of the Blood and Its Toxicity to Lupinus Albus Seedlings, J. Biol. Chem. **63**:LX, 1925.

toxicity in 27 depressed patients. They found that there was a relationship between the toxicity and the undetermined nitrogen of the blood and that the more severe the depression the more marked was the increase in toxicity and undetermined nitrogen. This increase in the toxicity of the blood of psychotic patients was confirmed by Herz and Weichbrodt [6] and by Tscherkes and Mangubi.[7] Herz and Weichbrodt found that blood serum obtained from patients with psychomotor disturbances gave a greater inhibition than that from patients without such disturbances, regardless of the diagnosis. Tscherkes and Mangubi tested the blood serum of 41 schizophrenic patients and found a constant and definite increased toxicity almost as great as that found in cases of pernicious anemia.

Our investigations were well under way when we learned of the work of Tscherkes and Mangubi. A careful study of their methods revealed the same objections as would apply to those of Macht, namely, a wholly insufficient number of tests per sample and an insufficient amount of control material. We therefore proceeded with our original plan of investigation, which was organized in such a way that the results could bear statistical analysis.

MATERIAL AND METHODS

This study is based on the results found in the phytotoxic tests completed on 68 male schizophrenic patients, who, as previously noted,[1] were for practical purposes representative of the average male schizophrenic population in the average state institution. Special care was taken to make sure that we were dealing with cases of schizophrenia in contradistinction to other psychoses with a schizoid coloring. The diagnosis in each case represented the unanimous judgment of three psychiatrists as well as of two internists and two psychologists who are experienced in dealing with psychotic patients. A further selection was made in that all the patients suffering from organic disease recognizable by careful physical and laboratory examinations were excluded as a routine. A few patients were dismissed from the group after a preliminary period because of intractability. No data from this group are included in the present report.

For control purposes we carried on simultaneous tests on 25 normal male physicians, medical students and laboratory workers who resided in this institution and were likewise free from any perceptible organic disease.

The seeds of Lupinus albus were used exclusively; they were purchased from two different supply houses. The seeds were soaked in tap water for twenty-four hours and then planted, hilus downward, in finely ground and moistened Sphagnum moss. All the work was carried on in a dark room, light being used only for the technical procedures involved. The temperature of the room was kept as consistently as possible around 20 C. The seedlings were allowed to grow (usually for seventy-two hours) until a length of root between 2 and

6. Herz, E., and Weichbrodt, R.: Die Toxicität des Serums und ihre Deutung, Deutsche med. Wchnschr. **50**:1210, 1924.

7. Tscherkes, L. A., and Mangubi, M. I.: Phytotoxische Eigenschäften des Blutes bei Schizophrenie, Ztschr. f. d. ges. Neurol. u. Psychiat. **132**:815, 1931.

3 cm. was obtained. They were then washed clear of the moss in tap water, and the initial length of the root as measured with a centimeter rule was recorded. Between 20 and 25 seedlings were used as a series for each test, including the Shive[8] nutrient fluid controls. With each day's experiments, control series were used as well as series of tests with solutions of urine and blood from the patients and normal subjects. Blood was obtained in the morning before breakfast, after a fifteen-hour fast, by venipuncture in the cubital space of the forearm. The blood was defibrinated by being shaken with sterile glass beads in a small sterile bottle of hard glass. The defibrinated whole blood was kept on ice until just prior to use, when the dilutions were made. The nutrient control fluid was a 50 per cent Shive solution which was also used as the diluting solution. The defibrinated whole blood was diluted to a 2 per cent solution. The total twenty-four-hour output of urine was diluted with tap water to a volume of 10 liters. This well mixed, diluted urine was then converted into a 2 per cent solution. The twenty-four hour urinary output was collected in a clean glass carboy with the addition of 1 cc. of toluene as a preservative and kept on ice. Small test tubes of hard glass (in upright racks) were filled to the brim with the test solution and one seedling put, root downward, into each test tube. The seedlings were evenly distributed so that the average root length of each series, to start, varied not more than 0.1 mm. from other series run on the same day. The seedlings were allowed to grow in the solutions in the dark for a full twenty-four hours and the length of the root was again measured and recorded.

The phytotoxic index of the test fluid was calculated by dividing the average twenty-four-hour gain in length of the roots in the test solutions by the average gain in length of the roots in the nutrient control medium. It can be expressed by the following formula:

$$\text{P. I.} = \frac{1' - 1}{L' - L} = \frac{\text{average 24 hour gain in length (mm.) of the test seedlings}}{\text{average 24 hour gain in length (mm.) of the nutrient seedlings}}$$

where 1 = average initial length of the roots in test solution (total length of the roots ÷ number of seedlings)

$1'$ = average final length of the roots after 24 hours in test solution

L = average initial length of the roots in nutrient solution alone

L' = average final length of the roots in nutrient solution alone

RESULTS

The results are summarized in the table.

Phytotoxic Index; Constants of Distributions

	Solution	Number of Cases	Minimum	Maximum	Range	Mean	Standard Deviation	Coefficient of Variation
Blood								
All patients	2%	87	0.41	0.79	0.38	0.590 ± 0.010	0.093 ± 0.007	16
Acute cases only	2%	8	0.42	0.77	0.35	0.574 ± 0.041	0.115 ± 0.029	
Chronic cases only	2%	79	0.41	0.79	0.38	0.594 ± 0.010	0.090 ± 0.007	15
Normal subjects	2%	21	0.47	0.84	0.37	0.606 ± 0.025	0.116 ± 0.018	19
Patients	1%	125	0.50	1.30	0.80	0.853 ± 0.014	0.157 ± 0.010	18
Normal subjects	1%	13	0.43	1.10	0.67	0.836 ± 0.055	0.189 ± 0.037	23
Urine								
All patients	2%	106	0.15	1.20	1.05	0.578 ± 0.018	0.186 ± 0.013	32
Acute cases only	2%	10	0.27	0.94	0.67	0.529 ± 0.064	0.203 ± 0.045	
Chronic cases only	2%	96	0.15	1.20	1.05	0.584 ± 0.019	0.183 ± 0.013	31
Normal subjects	2%	25	0.39	0.84	0.45	0.607 ± 0.023	0.114 ± 0.016	19

8. Shive, J. W.: A Study of Physiological Balance in Nutrient Media, Physiol. Researches **1**:327, 1915.

Blood.—Two per cent solutions of blood of 62 male schizophrenic patients were tested. Twenty-five of these had retests at different times, making a total of 87 tests in the series. Solutions of equal strength from 21 normal male subjects were also tested. No significant difference in toxicity was found between the two groups.

One per cent solutions of blood from 68 patients, as used by Macht and Tscherkes, were tested. Of these, 57 were retested, making a total of 125 different tests in the series. Solutions of blood of an equal strength from 13 different normal subjects were likewise tested. No differences in toxicity were found.

Urine.—The phytotoxic indexes of a 2 per cent solution of diluted urine were obtained on 68 patients, 38 of whom had retests, making a total of 106 tests in the series. Solutions of urine of equal strength from 25 normal subjects were also tested. No differences in toxicity were found.

Many investigators in this field of psychosis believe that the toxin is present or demonstrable only during the acute and not during the chronic stages. In other words, the toxin is demonstrable only early in the psychosis and not in the later stages. The mental symptoms persist even in the absence of the original etiologic agent. With this in mind we divided our group of patients into two classes, those with the more acute psychosis and the others in the more chronic stages. As indicated in the table, there were no significant differences between the two groups with the 2 per cent solutions of either blood or urine.

By statistical analysis we found no correlation between the phytotoxic index of the solutions of blood or urine of the patients and their age, period of hospitalization or subtype of dementia praecox.

COMMENT

The departure from Macht's technic (that of using from 5 to 10 seedlings for each test series) was made in an attempt to obtain more uniform results that might be accepted as valid by statisticians. If a reacting substance is found in varying amounts in a small number of tests it necessarily follows that this reacting substance can be measured quantitatively as of a mean strength if a sufficiently large number of tests are made. In our work it was soon apparent that any small number of tests could be selected and their results analyzed to give widely divergent findings. It was thought, then, that only the large series (20 tests or more) should be used as a basis for conclusions. By the same token, we used a 2 per cent strength of test solutions rather than 1 per cent as advised by Macht and his followers. It was thought that if a reacting substance is present in a 1 per cent solution, the results of it should be more uniformly obtained with a stronger solution. We changed

Macht's technic further by distributing the seeds in such a manner that the average initial root lengths of the series tested on any one day were, for practical purposes, equal. The average initial root length of any series used was always between 2 and 3 cm.

Defibrinated whole blood and blood serum were found to have the same phytopharmacologic inhibitory effect,[9] and we also demonstrated this to our own satisfaction in a number of cases.

Although no abnormal phytotoxin was observed in the blood, it was thought that it might be demonstrable in the urine. The diluting of the total twenty-four-hour urinary output to 10 liters put all the specimens on an equivalent basis. Ten liters was an arbitrary amount to which all twenty-four-hour outputs could be easily diluted. (Some of the patients excreted more than 5 liters.) It might be supposed that the addition of toluene to the urine was the sole cause of the inhibitory effect on the seedlings. We tested the toxic effect of different strengths of solutions of toluene in tap water and found that strengths up to four times greater than that used in our specimens had no toxic influence.

One per cent solutions of normal human blood inhibit to a slight degree the growth of the roots in twenty-four hours. It has been recorded that this phytotoxic index is 75 per cent.[10] The mean of the phytotoxic indexes of a 1 per cent solution from our normal subjects gave a higher figure, namely, 0.836, but the mean of the phytotoxic indexes of a 1 per cent solution of blood from our patients gave a figure equally high, 0.853. With the 2 per cent solutions of blood, the patients and the normal controls showed an almost identical mean phytotoxic index and range. This is contradictory to the findings of Tscherkes and Mangubi, who found a most pronounced toxic effect in schizophrenia, in some cases simulating the findings obtained for patients with pernicious anemia. In their method, however, there are several possible sources of error.

Even though the range of the phytotoxic indexes of the 2 per cent solutions of urine of the patients was more than twice as great as for the normal subjects, the mean phytotoxic index of both groups was strikingly the same.

SUMMARY

The inhibitory effects of 1 and 2 per cent solutions of defibrinated whole blood in a 50 per cent solution of nutrient Shive solutions on the

9. Macht, D. I., and Anderson, W. T. Jr.: Clinical and Experimental Studies on Phototherapy in Pernicious Anemia, J. Pharmacol. & Exper. Therap. **34**:365, 1928.

10. Macht.[4] Looney and Macht.[5] Herz and Weichbrodt.[6] Tscherkes and Mangubi.[7] Macht and Anderson.[9]

growth of the roots of Lupinus albus seedlings were studied simultaneously, using male schizophrenic patients and normal male subjects, all free from any perceptible organic diseases. The technic used was a modification of Macht's original procedure. From 20 to 25 seedlings were used in each series. An equal number of tests were made simultaneously, using nutrient solutions containing the same concentration of Shive medium as for each day's experiments. With each day's experiments the average initial lengths of the roots of each series were equal. The average initial root length of any series used was kept between 2 and 3 cm. A total of 68 patients and 21 normal subjects was thus tested; some of these had 1 or more retests.

A 2 per cent solution of diluted urine was likewise tested. In each instance the total twenty-four-hour urinary output was diluted to 10 liters. One cubic centimeter of toluene was added to each specimen as a preservative. For this study a total of 68 patients was tested, many of whom had one or more retests, and 25 normal subjects were likewise tested.

No significant differences were found in the inhibitory action of whole blood—in strengths of either 1 or 2 per cent—between male schizophrenic patients and the normal male controls.

Likewise no abnormal inhibitory action on the growth of these seedlings was demonstrable by using a 2 per cent solution of diluted urine.

No inhibitory action was obtained with toluene in solutions four times as strong as that which had been used as a preservative.

No existing correlation was found with the phytotoxic indexes of the solutions of blood or urine of our patients and their age, period of hospitalization or subtype of schizophrenia.

CONCLUSIONS

Neither the blood nor the urine of male schizophrenic patients showed any abnormal phytotoxicity demonstrable by the Lupinus albus test.

No significant differences in phytotoxicity were found between the more acute cases and the chronic cases with either blood or urine.

Clinical Notes

ENCEPHALOMYELITIS AND "HEMORRHAGIC VIRUS PNEUMONIA" COMPLICATING MEASLES

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The occurrence of lesions in the central nervous system as a complication of measles was first indicated by Lucas [1] in 1790, when he reported the clinical aspects of a case in which myelitis developed six days after the appearance of the rash. The condition was called "acute disseminated encephalomyelitis" by Westphal [2] in 1872. The pathologic changes were accurately described by Barlow and Penrose [3] in 1886. Individual cases have occasionally been reported in France, Germany and England during the last century, but in the past few years epidemics of measles have occurred in which the incidence of nervous sequelae is unusually high. During 1926 Neale and Applebaum [4] saw eight cases in the state of New York. Greenfield,[5] of London, reported having examined material from four cases within three months, in 1928. A report of the pathologic changes in four fatal cases occurring in the spring of 1930 in the city of New York was published by Ferraro and Scheffer.[6] A moderately severe epidemic of measles occurred in St. Louis during the late winter and spring of 1931-1932. Within a few months we saw four cases with nervous sequelae. Two patients recovered; of the two fatal cases only one came to autopsy.

REPORT OF CASE

Clinical History.—E. M., a white boy, aged 8 years, was admitted to the St. Louis Children's Hospital on Feb. 12, 1931, in a state of unconsciousness. At the age of 7 he had had whooping cough, and one month before admission an attack of catarrhal jaundice associated with anorexia and vomiting which lasted for three weeks and cleared up spontaneously. A week before admission there developed fever, conjunctivitis and catarrh; two days later the typical rash of measles appeared. The illness ran a normal course until twenty-four hours before admission, when the patient suddenly became delirious. After a short time the delirium

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1. Lucas, J.: London M. J. **11**:325, 1790, quoted by Greenfield.[5]

2. Westphal, C.: Ueber eine Affection des Nervensystems nach Pocken und Typhus, Arch. f. Psychiat. **3**:376, 1872.

3. Barlow, T., and Penrose: On a Case of Early Disseminated Myelitis, Tr. Med.-Chir. Soc. **70**:77, 1886.

4. Neale, J. B., and Applebaum, E.: Encephalitis Associated with Measles, J. A. M. A. **88**:1552 (May 14) 1927.

5. Greenfield, J. G.: The Pathology of Measles Encephalomyelitis, Brain **59**:171, 1929.

6. Ferraro, A., and Scheffer, I. H.: Encephalitis and Encephalomyelitis in Measles, Arch. Neurol. & Psychiat. **25**:748 (April) 1931.

disappeared, the patient became drowsy, and a stupor developed which deepened progressively.

On examination, shortly after admission, the boy was comatose and cyanotic. The temperature was 41 C. (105.8 F.) and the pulse rate 140. The respirations were noisy and rapid. The tongue was dry and coated, and the tonsils were reddened. Diffusely scattered râles were heard over both lungs. Both ear drums were injected, and the right one bulged. The heart sounds were normal. The abdomen was distended, and the fundus of the bladder was midway between the symphysis and the umbilicus. The lower border of the liver was palpable 2 cm. below the costal margin.

The pupils were small, equal and regular and reacted to light. The conjunctival and corneal reflexes were present. There was no obvious strabismus. The muscles of mastication appeared to be in tonic spasm, but if sufficient force was used the mouth could be passively opened against this resistance. The upper intercostal muscles and the accessory muscles of respiration were functioning. The diaphragm was not paralyzed, but there was flaccid paralysis below the level of the fifth dorsal segment. Both arms were spastic and flexed. The arm jerks were equal. The abdominal, cremasteric and plantar reflexes were absent, and the knee and ankle jerks were not obtained. There was incontinence of urine as well as of feces. The urine contained a trace of albumin, but was otherwise normal. There were 4,820,000 red blood cells and 17,400 white cells per cubic millimeter; of the latter, 74 per cent were neutrophilic leukocytes and 26 per cent lymphocytes. The tuberculin and Wassermann reactions were negative. A lumbar puncture yielded a clear fluid under increased pressure, which contained 180 cells per cubic millimeter, of which 79 per cent were lymphocytes and 21 per cent polymorphonuclear leukocytes.

The patient was placed in an oxygen tent and given dextrose solution intravenously. The cyanosis was relieved, but the respiratory rate did not change. The symptoms became progressively worse and the patient died forty-eight hours after admission.

The clinical diagnosis was encephalomyelitis following measles, secondary bronchopneumonia and acute otitis media.

Pathologic Examination.—Postmortem examination a few hours after death revealed the following changes:

Nervous System: The intracranial sinuses contained only fluid blood. The blood vessels of the meninges were engorged, and on the cut surface the vessels were distended. Many small petechial hemorrhages were visible in the white matter of both hemispheres and in the cervical cord.

After removing the brain and spinal cord small pieces about 7 mm. thick were cut from the cervical cord, the medulla, various parts of the cerebral cortex and the right basal ganglion. These were fixed in Zenker's solution, 5 per cent potassium bichromate solution or in 95 per cent alcohol. The rest of the brain and spinal cord was fixed in 10 per cent solution of formaldehyde containing 1 per cent sodium chloride.

A variety of stains was used: scarlet red for fat, Bielschowsky's stain for neurofibrils, Hortega's stain for microglia and toluidine blue for Nissl bodies. Tissue embedded in pyroxylin (celloidin) was employed for myelin sheath (Weigert-Pal, Anderson) and hematoxylin-eosin stains. Sections from material embedded in paraffin were stained with Mallory's eosin-methylthionine chloride (methylene blue), phosphotungstic acid-hematoxylin and connective tissue stains. Perdrau's method and van Gieson's stain were also used to demonstrate connective tissue fibrils.

The most conspicuous changes observed in the nerve tissue consisted of areas of perivascular demyelinization, which were scattered throughout the white substance of the brain, brain stem and spinal cord (fig. 1). They appeared to be most numerous in the central white substance at the level of the dorsal border of the corpus callosum and in the cervical cord (fig. 2). In hematoxylin-eosin sections they appeared as light staining circular or oval zones surrounding medium-sized or large veins.

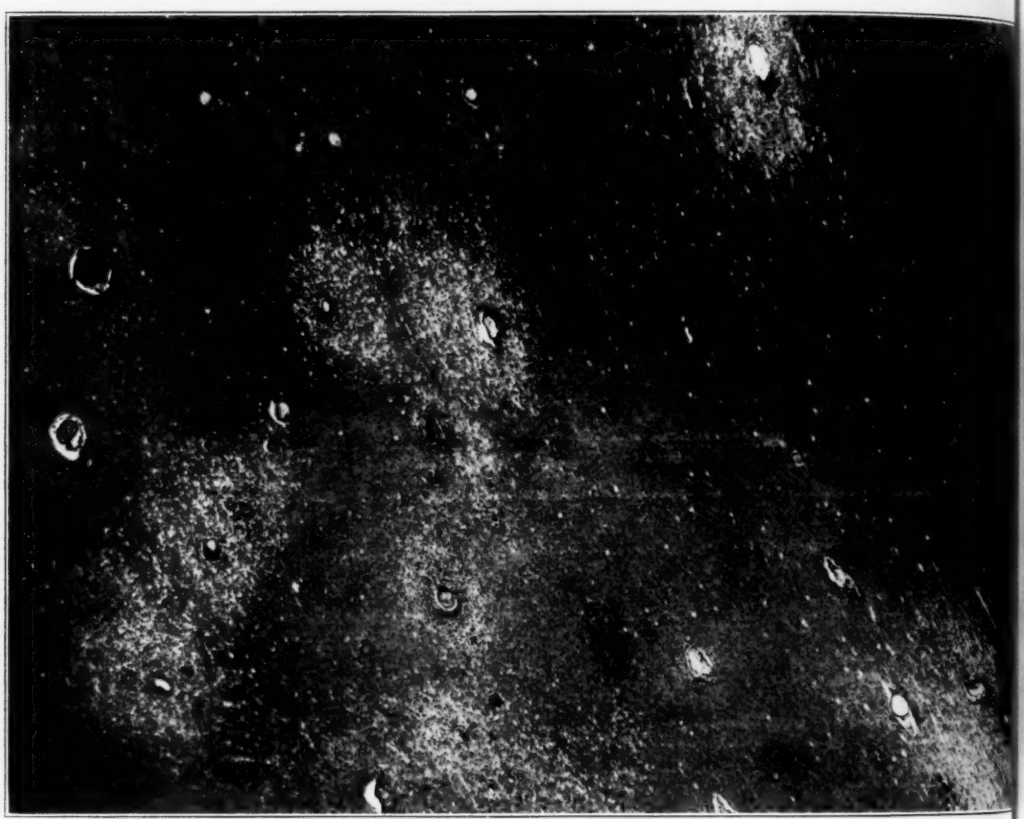

Fig. 1.—Low power photomicrograph of a section of central white matter showing numerous areas of perivascular demyelinization. The dark zones about several small vessels in the central portion of the picture represent areas of cellular infiltration. Phosphotungstic acid-hematoxylin stain.

In frozen sections stained with scarlet red the demyelinated areas did not show the general light brown staining that was evident wherever the myelin was undamaged. Surprisingly few fat globules staining red were present in any of the sections. However, a few large mononuclear cells, either in the perivascular lymph spaces or near the center of a demyelinated area contained fat globules in their cytoplasm.

In Weigert-Pal preparations (figs. 2 and 3) the transition from destroyed to normal myelin usually was not sudden; there was an intermediate zone in which

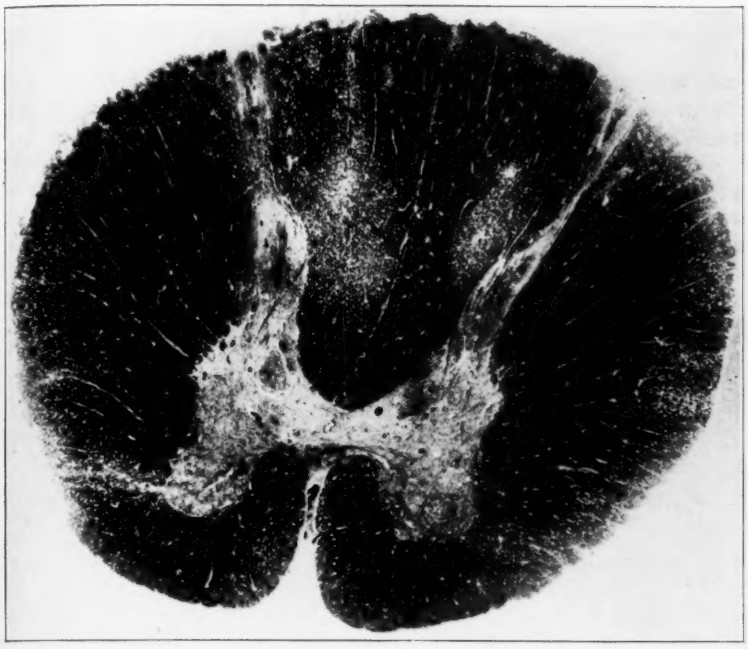

Fig. 2.—A section of the cervical cord stained by Weigert's method. Two areas of demyelinization can be seen between the posterior horns.

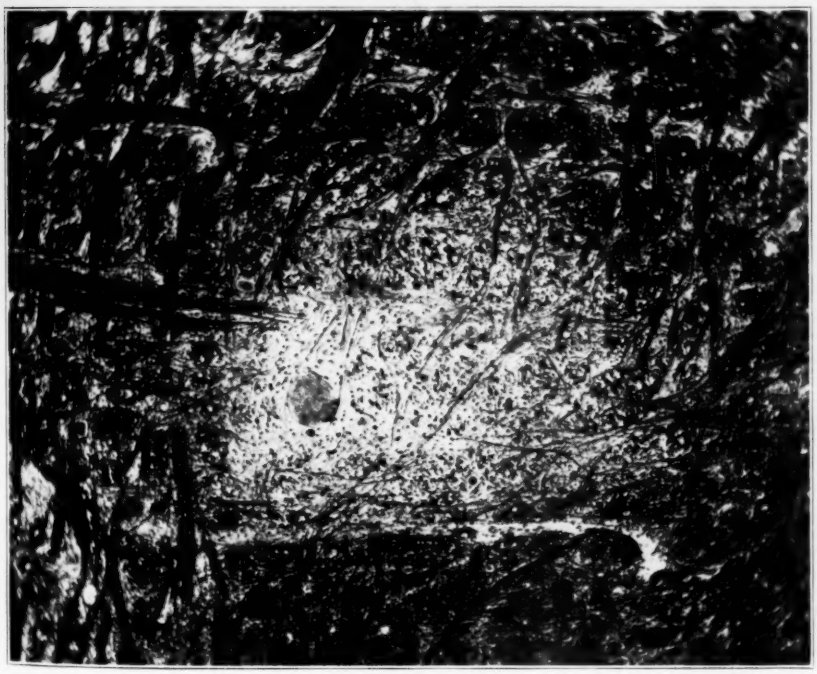

Fig. 3.—Higher power photomicrograph of the perivascular zone of demyelinization in the pons. Stained by Weigert's method.

the myelin sheaths stained lightly and were often thin or beaded. In the smaller patches these poorly stained or beaded myelin sheaths frequently swept across the blanched areas, at times very close to the central vessel.

Bielschowsky's method of impregnation with silver disclosed practically normal nerve fibers in the smaller areas of demyelinization. In the larger areas the nerve fibers situated at the periphery and in the outer half of the area appeared to be normal, while those close to the central vein often did not stain at all; in the intermediate zone they stained irregularly and occasionally showed circular or oval

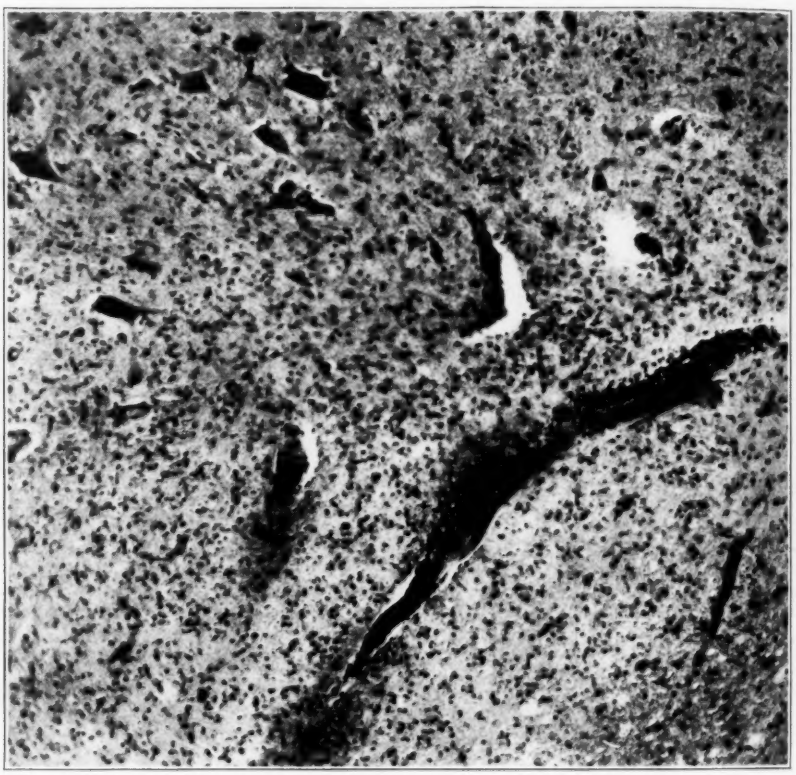

Fig. 4.—Low power photomicrograph of a portion of the anterior horn from a section of the cervical cord. A cuff of round cells almost encircles the longitudinally cut vessel in the lower right-hand part of the picture. There is also a general cellular infiltration throughout most of the section. Hematoxylin and eosin stain.

swellings. No abnormalities of arrangement were observed in the intracellular neurofibrils of the cortical cells or in those of the basal ganglia.

The demyelinated tissue was infiltrated with many mononuclear cells and a few polymorphonuclear leukocytes. This was always most marked in the zone around the central vein. Some of the mononuclear cells had light staining oval nuclei resembling the nuclei of astrocytes. The majority, however, had densely

stained nuclei exhibiting a variety of shapes and either no definite cytoplasm or a poorly defined cell outline. With Hortega's stain the bodies of these cells seemed to consist of loose, granular masses containing a dark nucleus; they are therefore regarded by us as microglia cells. Sections stained for fibrillar glia did not show an increase of this element in the demyelinated areas. Cuffs of mononuclear cells within the Virchow-Robin spaces were found around only a few vessels, although isolated cells or groups of cells were occasionally seen in these perivascular lymphatic vessels (fig. 4).

Other less specific changes included congestion of the blood vessels and degenerative processes in some of the nerve cells of the cortex, spinal cord and basal ganglia. Most of the small blood vessels were intensely engorged, especially the

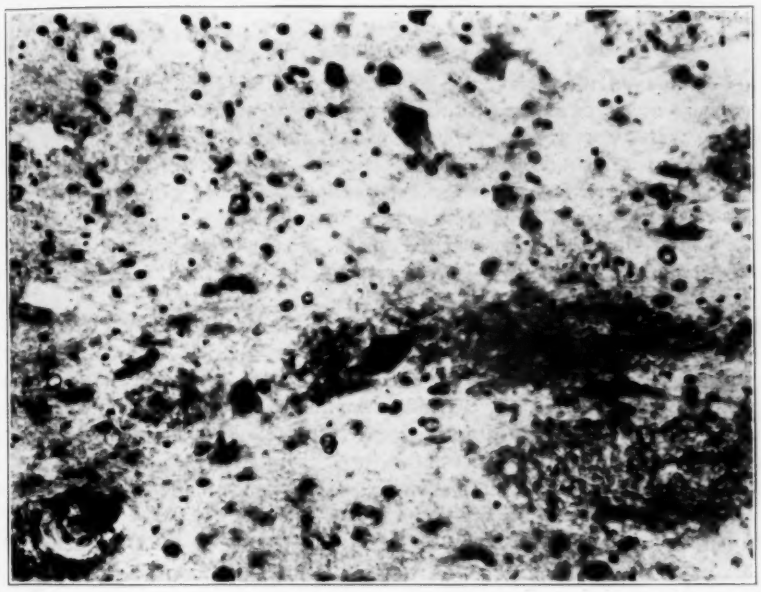

Fig. 5.—High power reproduction of a field from the anterior horn of the cervical cord, showing hemorrhage and cellular infiltration. Two areas of hemorrhage can be seen in the lower right-hand portion of the picture, and a little below the center is a ganglion cell completely surrounded by hemorrhage. Hematoxylin and eosin stain.

veins of the white matter. Those of the cortex were also congested though to a less extent. In a few places hemorrhages had occurred into the perivascular lymphatic spaces, and occasionally red cells were seen in a narrow zone of brain tissue surrounding the vessel (fig. 5). Some nerve cells adjacent to an area of demyelinization showed absence of Nissl granules, some chromatolysis and others pyknosis. Groups of small round cells surrounded and invaded some of the damaged nerve cells.

Lungs: Both showed small patches of red and gray consolidation. Areas of hemorrhagic consolidation were scattered through both upper lobes, and in smaller number in the lower lobes. Microscopically the red patches proved to be necrotic tissue in which blood had escaped into the alveoli and bronchioles. The lesions

resembled the hemorrhagic consolidation seen in epidemic influenza, and also the changes in hemorrhagic virus pneumonia which we have produced in animals with vaccine virus.[7] The gray patches were areas of lobular pneumonia. Large clumps of gram-positive cocci were found in sections stained for bacteria. In many patches the mucosa and often the entire bronchial wall, with the exception of the cartilage of the larger bronchi, were necrotic. In many places the lymphatic vessels were filled with coagulated fluid containing cells, fibrin and clumps of bacteria. No marked thickening or cellular infiltration of the bronchial or alveolar walls was found.

Liver: Small focal necroses, which as a rule occurred near the periphery of the lobule, were numerous.

Kidneys: There was cloudy swelling of the epithelial cells. Other organs showed no relevant changes.

COMMENT

The lesions of the central nervous system described here are similar to those reported as occurring in encephalitis following measles. Perivascular demyelinization, the most outstanding pathologic change in the majority of recorded cases, formed the most conspicuous lesion in our case. The perivascular cuffing with mononuclear cells, however, has not been as consistently present. It was not found by Brock[8] or Wohlwill,[9] and either was limited to one or two vessels, as in the case of Mosse,[10] or was patchy in distribution, as described by Bergenfeldt.[11] On the other hand, the cuffs of cells were a prominent feature in the case reported by Greenfield.[5] In our case this lesion was unimportant; it occurred around only a few of the veins, which showed collections of the small round cells in the Virchow-Robin spaces, completely encircling the vessel. These perivascular infiltrations were not present in regions in which demyelinization was extensive. Occasionally a patchy form of this infiltration, in which one or two small, isolated groups of cells were present in the perivascular lymphatic space, was found. Similar extra-adventitial cellular infiltrations in the perivascular zones of demyelinization have been reported in the majority of instances. Congestion and small hemorrhages about the blood vessels have been the most constant lesions and have been observed in almost all reported cases.

Two views concerning the etiology of acute disseminated encephalomyelitis are current: (1) a distinct disease caused by a specific neurotropic virus, or (2) a cerebral complication due to a generalization of the virus of the antecedent disease. The marked similarity between the pathologic lesions of measles and postvaccinal encephalomyelitis, as well as their resemblance to those in the encephalitis that occasionally follows smallpox and influenza,[12] is considered to support the view

7. Muckenfuss, R. S.; McCordock, H. A., and Harter, J. S.: Study of Vaccine Virus Pneumonia in Rabbits, Am. J. Path. **8**:63, 1932.

8. Brock, J.: Ueber Systematik und Klinik meningoencephaler Krankheitszustände im Kindesalter, Ztschr. f. Kinderh. **40**:552, 1926.

9. Wohlwill, F.: Ueber encephalomyelitis bei Masern, Ztschr. f. d. ges. Neurol. u. Psychiat. **112**:20, 1928.

10. Mosse, K.: Zwei Fälle von akuter Gehirndegeneration nach Masern, Jahrb. f. Kinderh. **112**:272, 1926.

11. Bergenfeldt, E.: Myelitis als Masernkomplikation, Acta med. Scandinav. **61**:281, 1924.

12. Ministry of Health: Report of the Committee on Vaccination, London, His Majesty's Stationery Office, 1930.

that encephalomyelitis is a disease in itself and not an unusual complication of the original disease due to a cerebral dissemination of the specific virus. This idea was also advanced by Perdrau [13] on the basis of experimental results. He showed that it is possible simultaneously to transmit two different viruses, such as those of herpes and vaccinia, through a series of rabbits, and to separate the two viruses by inoculating them into animals that have previously been immunized to one of the viruses.

However, as most of the diseases that precede acute disseminated encephalomyelitis are caused by filtrable viruses, at first thought it seems reasonable to ascribe this complication to the action of the specific virus of the antecedent disease. According to this view a generalization of the virus probably occurs in cases that are complicated by encephalitis.

The pulmonary lesions in our case are similar to those in other virus diseases and to the virus pneumonia which we have experimentally produced in animals with vaccine virus.[14] This similarity might be looked on as evidence of a possible generalization of the virus of measles, thus causing pneumonia as well as encephalitis, but this is not a necessary conclusion, because we have shown that the pulmonary lesions associated with or caused by different viruses are not type specific and cannot, therefore, be distinguished from one another. If acute disseminated encephalomyelitis is a disease entity and not a cerebral complication of the antecedent disease, the specific virus of encephalomyelitis may be looked on as the cause of the "virus pneumonia" as well as of the encephalitis. However, this interpretation is inadmissible, because interstitial bronchopneumonia and other virus-like lesions often occur in the lungs after measles and other virus diseases, although no signs or lesions of encephalitis can be discovered.

If this type of encephalitis were caused by the virus of the antecedent febrile disease one would expect rabbits inoculated with material from cases of postvaccinal encephalitis to show vaccinal lesions, and it should be possible to recover vaccine virus from these animals since they are susceptible to this virus; but negative results from such experiments have been recorded by Perdrau,[13] Maitland,[15] Kraus [16] and others. It therefore seems unlikely that the encephalitis following vaccination is due to vaccine virus.

The apparent epidemic nature of the encephalomyelitis complicating vaccination, measles and other exanthematous diseases is most easily accounted for by the theory that the condition is caused by a specific virus and not by the virus of the preceding exanthem. This specific virus must be widespread throughout the population during the years when the incidence of encephalomyelitis is high, but its presence is probably of little significance unless an attack of one of the exanthematous diseases, or some other condition, lowers the patient's resistance to this virus. All the evidence seems to favor such a view.

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13. Perdrau, J. R.: The Histology of Postvaccinal Encephalitis, J. Path. & Bact. **31**:17, 1928.

14. McCordock, H. A., and Muckenfuss, R. S.: Similarity of Virus Pneumonia in Animals to Epidemic Influenza and Interstitial Bronchopneumonia in Man, Am. J. Path. **9**:221, 1933.

15. Maitland, C., in Ministry of Health: Report of the Committee on Vaccination, London, His Majesty's Stationery Office, 1928.

16. Kraus, R.: Zur Aetiologie der postvakzinalen Enzephalitis, Wien. klin. Wchnschr. **40**:185, 1927.

Until it is possible to reproduce in animals the encephalomyelitis complicating measles, vaccination and occasionally other exanthematous conditions, the exact nature of the virus or viruses which cause this condition cannot, in all probability, be definitely determined.

SUMMARY AND CONCLUSIONS

A case of encephalomyelitis following measles and "hemorrhagic virus pneumonia" is described.

The pathologic lesions of the nervous system are described and compared with those which have been previously reported. In our case the perivascular cuffing was not a prominent feature.

The pulmonary lesions in this case were similar to those in other diseases caused by viruses and in experimental virus pneumonia.

EXTENSIVE LONGITUDINAL CAVITATION OF THE SPINAL CORD ASSOCIATED WITH A CIRCUMSCRIBED INTRAMEDULLARY TUMOR

NATHANIEL J. BERKWITZ, M.D., MINNEAPOLIS

Medullary cavities associated with tumors of the spinal cord were reported by early pathologists and anatomists such as Etienne (1546), Brunner (1688) and Morgagni (1740).[1] It was not until 1824, however, that the term "syringomyelia" was introduced by Ollivier.[2] He thought that the mere presence of a patent central canal was evidence of an abnormal condition. At that time it was commonly believed that normally the central canal became extinct during the latter part of fetal life. In 1859, Stilling[3] was the first to establish definitely that the central canal is normally present, but he also believed that syringomyelia is always the result of dilatation of the central canal.

Because of different pathologic changes considerable interest and discussion arose as to the cause of syringomyelic cavities, and many theories were advanced in the latter part of the nineteenth century. It was later pointed out by Hoffmann,[4] Schlesinger[5] and Patoir and Raviart[6] that syringomyelia is not a nosologic entity, but rather a syndrome resulting from numerous causes, such as gliomas, developmental anomalies, trauma, hematomyelia and softening.

In 1881, Langhans,[7] reporting four cases of cavity formation in the spinal cord from nongliomatous tumors, was the first to demonstrate that circulatory stasis by compression from the tumor produced intramedullary cavities not connected with the central canal. Lhermitte and Boveri[8] were able to produce cavitation by introducing a strip of Laminaria within the spinal canal of dogs, and reported that cavities may result from pressure. According to them, the consequent circulatory stasis from pressure results in necrosis and softening, which extends along the spinal cord.

From the Division of Nervous and Mental Diseases, University of Minnesota Medical School.

1. Haenel, H.: Syringomyelia, in Lewandowsky, M. H.: Handbook der Neurologie, Berlin, Julius Springer, 1911, vol. 2. p. 572.

2. Ollivier, C.-P.: De la moëlle épinière et de ses maladies, ed. 2, Paris, Crevot, 1824.

3. Stilling, B.: Neue Untersuchungen über den Bau des Rückenmarks, Cassel, H. Hotop, 1859.

4. Hoffmann, J.: Zur Lehre von der Syringomyelie, Deutsche Ztschr. f. Nervenh. **3**:1, 1892.

5. Schlesinger, H.: Die Syringomyelie, ed. 2, Vienna, Franz Deuticke, 1902.

6. Patoir, J., and Raviart, G.: Gliomes et formation cavitaire de la molle, Arch. de méd. expér. et d'anat. path. **13**:93, 1901.

7. Langhans, T.: Ueber Höhlenbildung im Rückenmark als Folge von Blutstauung, Virchows Arch. f. path. Anat. **85**:1, 1881.

8. Lhermitte, J., and Boveri, P.: Sur un cas de cavité médullaire consécutive à une compression bulbaire chez l'homme et étude expérimentale des cavités spinales produites par la compression, Rev. neurol. **20**:385, 1912.

The following reports, relating to the presence of large intramedullary cysts associated with circumscribed intramedullary tumors of the spinal cord, have recently appeared. In Delafield and Prudden's textbook of pathology [9] a statement is made that " there seems to be a class of lesions of the cord, usually classed as syringomyelia, in which cavities of various forms coexist with a tumor in the vicinity of the central canal. But these cavities appear to be formed, not by breaking down of the tumor tissue, but in some other way as yet little understood."

Kirch [10] classified cavities of the spinal cord resulting from tumors into two groups. In one group he included cases in which the cavity is formed by the breaking up of centrally lying tumor tissue, so that the inner wall of the cavity is composed of remains of tumor tissue. In the other group he classed cavities which result from circulatory disturbance due to thickening of blood vessels and thromboses which, in turn, cause a serous transudation in the tissue around the tumor, forming an encapsulated glial cavity. He reported three intramedullary tumors of the spinal cord with syringomyelic cavities extending several segments beyond the tumor tissue. He believed that the size and the extent of the tumor were of no great importance. According to him, such a tumor may extend the entire length of the cord without the formation of a cavity, but with a tumor rich in blood supply cavities are formed frequently well beyond the tumor tissue. It was his opinion that occlusion of the vessels by endothelial proliferation and thrombosis produces vascular changes which create stasis, and that this in turn produces syringomyelic cavities.

Kernohan, Woltman and Adson,[11] in a review of intramedullary tumors of the spinal cord, found syringomelic cavities in five of ten cases in which autopsy was performed. However, they did not find a single case of cavity formation associated with any of their extramedullary tumors of the spinal cord. They did not attempt to offer an explanation for this frequent association of cavities with intramedullary tumors. In a recent communication, Dr. Kernohan informed me that nearly 80 per cent of the cases of intramedullary tumors studied to date at the Mayo Clinic showed syringomyelic cavities.

A small circumscribed intramedullary tumor of the spinal cord which was associated with an extensive intramedullary cyst was reported by Dowman and Smith.[12] They were unable to find any tumor cells in the cyst. They emphasized that the presence of a cyst in the spinal cord should suggest the possibility of a neoplasm above or below it. Recently, Davison and Keschner [13] reported two similar cases. They claimed that extramedullary or intramedullary expanding lesions may lead to cavity formation by: "(1) destruction of tissue of the cord

9. Delafield, F., and Prudden, T. M.: A Handbook of Pathological Anatomy and Histology, ed. 22, revised by F. C. Wood, New York, William Wood & Company, 1922, p. 1154.

10. Kirch, E.: Ueber die pathogenetischen Beziehungen zwischen Rückenmarksgeschwülsten und Syringomyelie, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**: 231, 1928.

11. Kernohan, J. W.; Woltman, H. W., and Adson, A. W.: Intramedullary Tumors of the Spinal Cord, Arch. Neurol. & Psychiat. **25**:679 (April) 1931.

12. Dowman, C. E., and Smith, W. A.: Intramedullary Cyst of the Spinal Cord Associated with a Circumscribed Intramedullary Tumor, Arch. Neurol. & Psychiat. **21**:583 (March) 1929.

13. Davison, C., and Keschner, M.: Myelitic and Myelopathic Lesions: VI. Cases with Marked Circulatory Interference and a Picture of Syringomyelia, Arch. Neurol. & Psychiat. **30**:1074 (Nov.) 1933.

by an intramedullary tumor; (2) circulatory interference within the cord from compression of the intraspinal vessels by an intramedullary tumor, or (3) circulatory interference by direct compression of the cord and its vessels by thickened meninges or by an extramedullary neoplasm or by both."

REPORT OF CASE

History.—M. P., a girl, aged 14 years, was admitted to the University Hospital on Jan. 6, 1930. Her first symptom was weakness in the right thigh which appeared on Nov. 27, 1929, while she was walking home from school. On the following day the weakness extended down the leg. A week later the other leg became involved. No pain was experienced during the first week, but at the beginning of the second week a sharp pain appeared suddenly in the spine in the lower thoracic region. The pain recurred intermittently and radiated in a girdle-like manner. Two weeks later the pain subsided for three days, and at this time the patient first noted numbness in the lower extremities, extending from the waist down. On Dec. 25, the pain reappeared in the scapular region and radiated anteriorly. She also began to have urinary retention at this time, so that it was necessary to catheterize the bladder daily. On Jan. 2, 1930, both knee reflexes were increased; a positive Babinski sign was obtained on the right side, and the patient was unable to flex or to extend the toes. On Jan. 4, the right knee reflex was absent. The spinal fluid obtained that day was clear, the Nonne-Apelt reaction was negative, and no cells were found. Cultures of the fluid were sterile.

Examination.—The patient was well nourished and well developed on the day of admission to the hospital. She cried because of intense pain in the thoracic region of the spine. The pulse rate and the temperature were normal. Neurologic examination disclosed no paralysis of the cranial nerves. Superficial sensation was impaired below the second thoracic segmental level, but variations in responses below this region suggested that some sensory fibers remained intact. Above this level no sensory changes could be elicited. The deep reflexes in the upper and lower extremities were absent. There was some decrease of motor power in the upper extremities, and the lower extremities were flaccid and completely paralyzed. The abdominal reflexes were absent. Positive bilateral Babinski signs were obtained, and a mass reflex occurred on stimulation of the soles.

Course in Hospital.—On Jan. 7, the patient was carried to the roentgen department for a roentgenogram of the spinal column. She lost motor power of the upper extremities rather suddenly while on the table. Shortly afterward the spinal fluid pressure was found to be 60 mm. of water. No response was elicited on jugular compression. The fluid was greenish yellow, and the reaction to the globulin test was strongly positive. There were 5 cells per cubic millimeter. The Wassermann reaction of the spinal fluid was negative, and cultures were again negative. The sensory level extended upward during that day from the second thoracic to the fourth cervical segmental level. Below this level, superficial and deep sensations were markedly impaired. In some areas sensation to touch was more preserved than that to pain and temperature, indicating that there was some dissociation and that the lesion was intramedullary. A cisternal puncture was performed on the next day, and clear fluid was obtained under 200 mm. of water pressure. There were 65 cells per cubic millimeter, 80 per cent polymorphonuclears and 20 per cent lymphocytes. The Nonne-Apelt and Noguchi tests gave strongly positive reactions. One cubic centimeter of iodized poppy-seed oil 40 per cent was introduced into the cisterna. Roentgenograms showed that

the oil gravitated to the sixth thoracic segmental level and remained there, indicating a total block. On the following day lumbar puncture was repeated, but only a few drops of cloudy fluid were obtained.

The sensory level of hypesthesia continued to ascend during the patient's third day in the hospital. By evening it had reached the second cervical segmental distribution. The temperature rose to 100 F., and the pulse rate increased to 110 per minute. The intercostal muscles became paralyzed and the respirations labored. It was necessary to institute artificial respiration at times during the night. On the following day the temperature rose to 106 F. by rectum. Respiration improved, and no definite change was noted until January 11, when the sensory level receded and the level of hypesthesia descended to the clavicle on the right and to the second rib on the left. No sensation was present in the right arm, but some sensation returned in the left. The patient breathed more freely. A slight amount

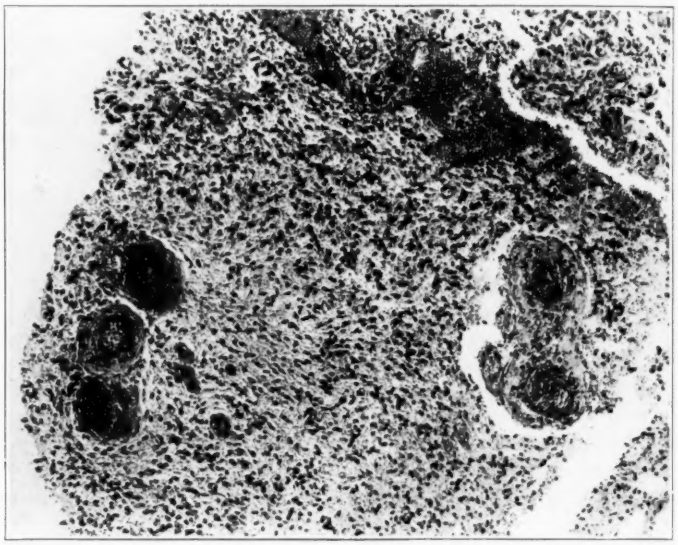

Fig. 1.—Photomicrograph of a section through the tumor, showing tumor cells, thrombosed vessels, hemorrhage and softening. Hematoxylin and eosin stain.

of motor power returned in the left forearm. On the next day there was some return of motor power in both upper extremities. There was some return in superficial sensation and vibration sense, and muscle pain sense was elicited in the lower limbs.

A week later the patient showed improvement and was more comfortable. She was able to raise the left arm, but could not move the fingers. The spinal fluid pressures in the lumbar region and cisterna were both about 80 mm. of water, and the response to jugular compression was normal, indicating that the spinal block had disappeared. There were 6 cells per cubic millimeter in the spinal fluid obtained from both lumbar and cisternal punctures and no increase in globulin was found. Further, roentgenograms showed that the block had disappeared, since most of the iodized oil had gravitated to the sacral region. The temperature, however, remained elevated between 100 and 106 F., and the pulse rate ranged from 90 to 100 per minute. A leukocytosis of 17,000 was present.

On January 24 it was observed that the sensory level continued to descend, and superficial sensation reappeared above the sixth thoracic segmental level on the left and the second thoracic segmental level on the right. A weak left biceps reflex was obtained. Practically no changes were elicited in the neurologic examination from that time on. Cystitis and pyelonephritis developed. The septic temperature continued, and trophic ulcers appeared over the trochanters and other bony

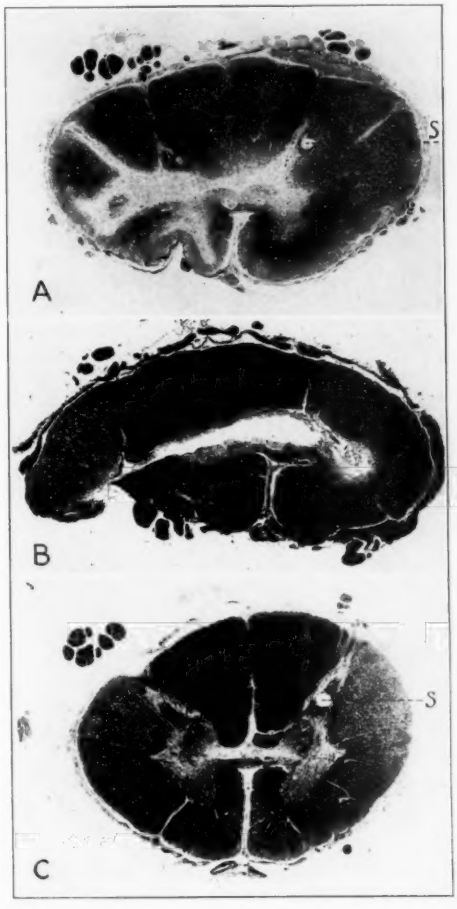

Fig. 2.—*A*, lower cervical region several segmental levels above the tumor, showing softening and cavitation, not communicating with the central canal. (The artefact *S* in this figure and in *C* was made by a needle to string the tissue for identification.) Pal-Weigert stain. *B*, a section through the lower thoracic region about three segmental levels below the tumor, showing softening and cavitation. Iron and hematoxylin stain. *C*, a section through the upper lumbar region, showing softening and cavitation in the left half of the posterior commissure, not communicating with the central canal. Pal-Weigert stain.

prominences. The patient began to lose weight rapidly, but showed improvement in sensation and motor power in the upper extremities. The general physical condition gradually grew worse, and death occurred on March 15.

Necropsy.—Grossly, the cord was swollen and the meninges were thickened in the midthoracic region. The blood vessels in this region were engorged. A rather doughy, white, well circumscribed tumor mass, about 4 cm. in length, practically replacing the tissue of the cord, was found at the fourth and fifth thoracic segmental levels. On sectioning the cord at various levels, an extensive cavity, containing some serous fluid, was found above and below the tumor, extending upward to about the fifth cervical segment and downward to the sacral region.

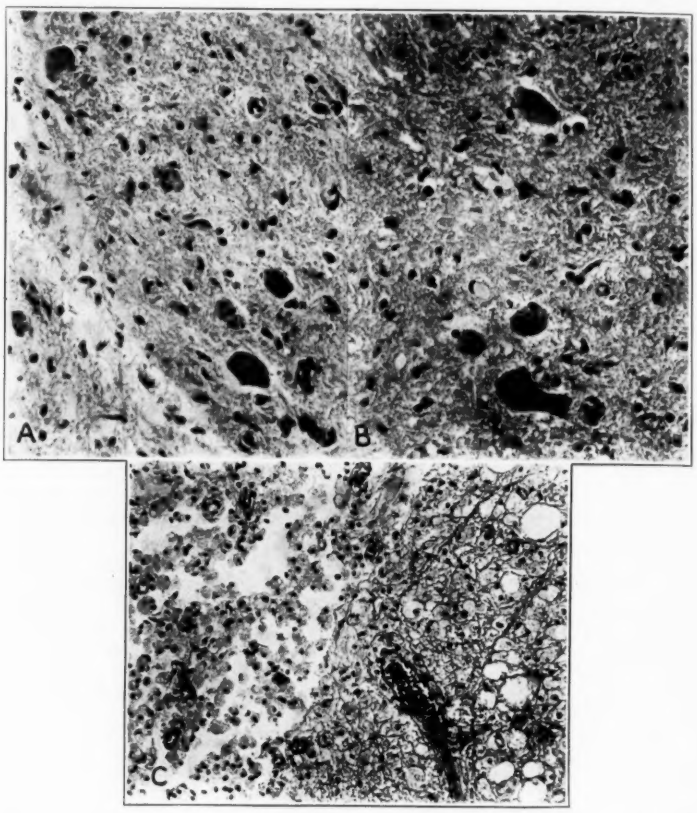

Fig. 3.—Photomicrographs of a section in the upper cervical region showing: *A*, chromatolysis of the right anterior horn cells and an increase of glial cells; *B*, less involvement in the left anterior horn; *C*, softening and numerous gitter cells, with swelling and degeneration of the myelin sheaths in the region of the posterior commissure. Hematoxylin and eosin stain.

Microscopic examination revealed a spongioblastoma multiforme with hemorrhage, increased vascularity, thrombosis and areas of softening. Practically all of the vessels within the tumor were thrombosed (fig. 1). No evidence of thrombosis was seen outside the tumor mass. Most of the tumor cells were irregular in outline, with large nuclei that were rich in chromatin material. They assumed various rudimentary formations and showed a tendency to cluster about blood vessels. A few giant cells were also present. Bielschowsky and Pal-Weigert

stains revealed that practically all of the axis-cylinders and myelin sheaths were destroyed and replaced by tumor tissue. No tumor cells were found along with the cavity above or below the tumor mass. The meninges surrounding and in close proximity to the tumor were greatly thickened. The meninges elsewhere were normal. Some glial proliferation was found lining the cavity adjacent to the tumor, but no definite lining of the cavity was seen beyond a few cord segments. The cavity did not communicate with the central canal, but dissected its way chiefly posteriorly in the gray matter (fig. 2).

Microscopic sections showed that the spinal cord in the upper cervical region was involved beyond the level where the cavity could be seen grossly. In this region, swollen and fragmented myelin sheaths were seen, and numerous fat granule cells (gitter cells) were present (fig. 3). The anterior horn cells and processes were swollen, and chromatolysis was pronounced. The nuclei were displaced or extruded, and Nissl substance was decreased or absent in most of the cells. As shown in figure 3, the right anterior horn was more involved than the left.

In the lumbar region the cavity was narrower, and only the left half of the posterior commissure was involved. The cavity did not communicate with the central canal (fig. 2 *C*). The anterior horn cells in the lumbar region were not involved, but there was a secondary degeneration of the descending tracts.

COMMENT

This case is of particular interest because it was observed during the period of transitory spinal block and probably during the course of the development of softening and cavitation, as suggested by clinical evidence. During the first few days following the patient's admission to the hospital the picture was more suggestive of an acute nonspecific ascending meningomyelitis than of tumor of the spinal cord. Both sensory and motor paralysis developed rapidly, ascending about eight segmental levels within a period of three days. There was some dissociation in sensation, indicating the presence of a central lesion. Because of the rapid course and the uncertainty in the diagnosis, operative intervention was not attempted. The ascent of the paralysis and the interval of arrest, followed by sensory and motor improvement from above downward, were difficult to account for at that time. At about the same time the patient's condition began to improve; the spinal fluid became normal, and the pressure of the fluid returned to normal. Death, however, occurred from toxemia, probably secondary to the pyelonephritis and extensive bed sores.

The tumor was a small, circumscribed spongioblastoma multiforme containing numerous thrombosed blood vessels. An irregularly shaped cavity extended several segments above and below the tumor mass. Microscopic sections revealed toxic changes in the remaining gray matter along the entire length of the cord above the sacral region, which showed only degeneration of the descending spinal tracts. As in the other cases reported, there was more involvement of the cord above the tumor than below it. Except for a mild reactive gliosis lining the cavity a few centimeters proximal to the tumor, no definite lining was seen. No tumor cells were found beyond the tumor mass.

This case differs from most cases reported in that no definite lining of the cavity was found. It may be that a lining would have formed by progressive glial changes had the patient lived longer. It is difficult to explain how a local vascular interference alone can produce an extensive cavity, since it is known that the vessels in the spinal cord anastomose at each vertebral level. From the clinico-

pathologic study of the case reported it appears possible, as Kirch suggested, that a marked sudden circulatory disturbance is the cause of necrobiosis, producing softening and cavitation of the gray matter in the spinal cord.

SUMMARY AND CONCLUSIONS

Cavities in the spinal cord are known to arise from various morbid conditions, suggesting that syringomyelia is not a nosologic entity. Such cavities may be found within intramedullary tumors of the spinal cord resulting from necrosis within the tumor. They may also be found outside the limits of the tumor. It is suggested that this condition may be secondary to necrobiosis associated with sudden impairment in circulation.

Differing from cases reported previously, this case showed no dense fibrous scar forming a wall of the cavity, presumably because the patient died before progressive glial changes had sufficient time to make their appearance.

News and Comment

AMERICAN COLLEGE OF PHYSICIANS

The American College of Physicians will hold its nineteenth annual clinical session in Philadelphia, April 29 to May 3, 1935.

SPECIAL ARTICLE

THE OBSESSIVE PARAPHILIAS (PERVERSIONS)

A CRITICAL REVIEW OF STEKEL'S WORKS ON SADISM, MASOCHISM AND FETISHISM [1]

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He who shows me a new plant pleases me more than all the dialecticians who subtilize probabilities. For what is their philosophy? An opinion concerning opinion. —*Helvetius.*

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INTRODUCTION

Among the many aberrant forms of sex behavior which come to the attention of the psychotherapist is that displayed by a type of patient who, although claiming a thoroughly normal attitude in matters of sex, is, for reasons unknown to him, greatly perturbed by many sexual problems. These patients say that their mind dwells continually on women, which they characteristically designate as mental masturbation, but try as they may they are unable to effect a normal relationship. Excessive masturbation is universal among them and is a source of great worry to them. Such relationships as may be established between them and the opposite sex assume forms so grotesque and bizarre as to betray even

1. Stekel, W.: (*a*) Sadism and Masochism: The Psychology of Hatred and Cruelty. Authorized English Version by Louise Brink. Two Volumes. New York, Horace Liveright, Inc., 1929. (*b*) Sexual Aberrations: The Phenomenon of Fetishism in Relation to Sex. Authorized English Version by S. Parker. Two Volumes. New York, Horace Liveright, Inc., 1930.

superficially their abnormal character. Every possible relationship may be indulged in and all sorts of gratifications achieved, except one—the normal. Among these abnormalities, properly spoken of as paraphilias,[2] sadomasochism and fetishism occupy a conspicuous place.

It is striking that, although many of these patients beg for help, few actually come for treatment. Investigation into their histories shows that they have avoided opportunities to become normal. Their passion is described as physical love, and they are fond of asserting that they are not capable of any other love. If they find a desirable partner they soon begin their depreciation and arrange misunderstandings, quarrels, etc. In this respect they are much like other neurotic persons. For neurotic persons, as a group, seldom wish to get rid of their neurosis, but only of the distressing symptoms. The neurosis has developed largely as a protective mechanism against the inner menaces; it is a crutch for a crippled life, and the patients fear to lose the only support they know of.

In the four volumes under consideration, Stekel discusses in intimate detail the pathologic manifestations of loves and hates, which are most exquisitely expressed in the sadomasochistic and fetishistic types of neuroses. His conclusions are buttressed by a wealth of clinical material (nearly two hundred cases), some of these being among the most amazing to be found in the entire psychopathologic literature. They offer a remarkable contribution to the problem of erotic symbolism, and their richness can be appreciated only if they are read in the original. While appearing relatively rarely in pure forms, the reactions described contribute in a large share to neuroses and psychoses. Their wide and vicarious ramifications penetrate into many phases of one's personal and social life, and their consideration is, therefore, of practical, no less than clinical and theoretical, import.

The lay person whose sex life is normal will deny the existence of such cases; the general medical practitioner who, at best, has only a superficial glimpse of them, will minimize their importance. Only the practicing psychotherapist who has had the opportunity to probe below the anamneses obtained as a routine can appreciate fully how widespread these cases are and what a tremendous therapeutic problem they present. Almost universally these patients present a normal appearance, but the tremendous emotional cost at which this is bought is hardly appreciated. When they come for treatment they are often cooperative, but they soon exhaust the physician's patience and nearly drive him to despair. Objectively, they soon come to appreciate how silly it is to indulge in endless

2. The term commonly used for this is perversion, which is entirely undesirable, as it has attached to it a social stigma hardly acceptable in a scientific discussion. The term paraphilia adequately comprises all sexual activities that are beside the pale of the normal and do not lead to normal gratification. Within this group homosexuality should be spoken of as inversion, as suggested by Havelock Ellis.

day-dreaming; yet with all their intelligence, they cannot effect the least control of the situation. One is dealing here with a remote and dark corner in the domain of psychopathology, with problems that are at once bizarre and baffling. While they seem insignificant to others, to the psychotherapist they are glaringly real. These patients are among the most intelligent one sees. Among them are friends and colleagues, for professional men and scientists contribute in no small proportion to this group. The material, especially the case material, presented in this study, represents but a small fraction of what one sees in practice; from this one may judge the great variety of forms of sex behavior which these patients present.

LOVE AND HATE

If one were to judge the importance of love by the number of books written about it or by the position which it occupies in religion and culture, the impression would be gained that it constitutes the central force of existence. However, a deeper insight into life teaches that it is hate which is the great motive power of all that happens. Man is essentially primitive and is ruled by impulses and instincts; that which opposes the satisfaction of instinct is looked on as hostile, and that which is hostile is hated. Thus, the primitive attitude of man is hatred; love is a cultural product. Such love as there is in primitive man is directed merely to his own ego, and one comes to love another when the other becomes a source of pleasure and comfort. If the pleasure is withdrawn, a feeling of deprivation accompanied by pain sets in, and hate makes its appearance. This is easily observed in the distress connected with the weaning of a child.

The readiness of mankind to hate is extraordinarily great and reveals itself in a variety of forms. Civilization compels either the renunciation or the concealment of this tendency to hate and its exchange for the readiness to love. The function of love is to retain that which is pleasant; the function of hatred is to remove the noxious object or stimulus. Accordingly, one may group around love and hate the large number of emotional reactions that depend on them, compassion, trust, friendship, kindness and affection being expressive of the sympathetic emotions and jealousy, disgust, rage, envy, etc., being expressive of the antipathic emotions.

Love and hatred are closely bound together and are genetically related. They reverse and displace each other, so that it may truly be said that there is no hatred without love. Loves and hates may be unconscious, and behind a conscious love or hate are often concealed the much more important love and hate objects of the unconscious.

Love and hate being expressions of one and the same impulse, there is observed in the partners a constant strife between the will to power and the will to submission. There is a relationship between

sexual pleasure and cruelty. Love-biting and efforts to squeeze, pinch or crush the partner suggest the connection and are not to be explained merely by the desire to make the partner feel one's power and to cause him pain. When there is a strong affective discharge, pain is not felt as pain, and the infliction of pain may serve only to increase the pleasure. That is to say, in affective tension pain is felt not as pain but as a nervous stimulus. Because pain and pleasure use the same pathways (stroking, if increased to blows, causes pain), the affective excitement changes the character of the pain and transforms it into pleasure. A streak of cruelty is found in the noblest of men. It is rarely indeed that another's pain leaves one entirely indifferent. The instinctive reaction to another's misfortune is that of malicious pleasure in the fact that it did not happen to oneself. Following this, as a compensation, there is often a feeling of compassion by which one experiences the other's pain as if it were one's own. Occasionally, the suffering of others creates in some a feeling of sexual satisfaction. Those who suffer envy others their happiness; suffering, they become cruel, enjoying another's hurt and delighting in a sense of power over another's life. But cruelty is essentially a product of civilization. Primitive man, while he was merciless in removing obstacles that stood in his way, was actually not cruel, for the element of pleasure in the act was absent. Cruelty is often toned with a large element of sexual pleasure. Religion, too, often has a sexual background, and religion and cruelty are closely related, as seen in the activities of the various sects. Many psychopathologists have emphasized that the normal sex impulse is always bound with a more or less prominent brutal element.

Ontogenetically, the child represents primitive man striving for permanent pleasure and therefore confronting the world with hostility. Through constant attention to its needs and comfort the mother slowly wins the love of the child and becomes a part of his ego; later, the father is also incorporated in the scheme. Thus the family becomes the school of love. But strangers are still feared and, being feared, are hated, for anxiety is fear of the pain which the stranger may cause. From the family, love is carried over to the community, which becomes the root of social sentiment (local, racial, national, etc.) and which in turn permits love and hatred to be transferred to other associated groups. It may thus be said truly that as one was in the family so will one be socially.

Without implanted conscience the child would not adjust himself to the laws of civilization. His hatred, like every hatred, is deadly, and accompanying it are death wishes, which are the first source of a consciousness of guilt (sin) that demands punishment. Through this sense of guilt an inversion of the hatred comes about, and all the hatred originally directed outward is now directed against the child's own ego. The consciousness that in self-punishment he erects a barrier to further

expressions of hate affords him pleasure. Thus one witnesses the strange psychic phenomenon that under certain circumstances allows the conversion of pain into pleasure, especially if it enters into association with the sexual impulse.

When a person loses his ability to love and to hate, there is a neurosis. This loss, however, is only apparent, for psychoanalysis in such cases reveals that love is already fixed on an object and cannot be brought into the light of consciousness because of the consorship. Because of the fact that it is essentially a manifestation of the instinctive life of the past, neurosis is a phenomenon of reversion. The further back the reversion goes the more pronounced is the attitude of hate and the less is the capacity for love. Thus, neuroses are essentially asocial, for love of others and obedience in the service of culture are fundamental to social feeling. In many neuroses one is able to demonstrate the presence of the sadistic hate component as a driving motive and its transformation into a consciousness of guilt and into suffering as self-punishment.

DEFINITIONS

Until the advent of psychoanalysis, sadism and masochism were regarded as two fundamentally different forms of sexual aberrations, although, even on superficial observation, the two are often found in the same person. Psychoanalysis shows this connection to be a matter of regular association of the two currents, the bipolar expression of one and the same energy, and the question as to which is primary and which secondary is more or less superfluous. This combined appearance of sadism and masochism is not incidental. Masochism is only a special form of sadism. Sadists manifest masochistic traits, all of which exhibit the character of penance. Masochism is more acceptable socially, while the primary sadism is much more hidden. Even though one hears of masochistic tendencies which go back to the earliest childhood, a longer analysis shows relentlessly behind every delight in one's own pain the inclination to atone for the pleasure in another's hurt.

The nature of the bipolarity is such that all reactions in the love life must manifest themselves negatively and positively. Fear and desire, disgust and longing, hate and love are the most familiar forms of expression. Bisexuality itself is the most eloquent manifestation of bipolarity.

The two fundamental forms in which sexuality may manifest itself are the will to power and the will to submission, and all psychic processes stand in the service of the two forces which, in a sense, maintain a balance. Social life has brought it about that in the canon of the normal the man tends more to the side of sadism, the woman to that of masochism. Man's love is domination, woman's submission. Where one finds the opposite, this is felt as unnatural.

Krafft-Ebing defined masochism as a peculiar perversion of the pyschosexual life. The person affected by it is dominated in his sexual feelings and thought by the idea of being completely and unconditionally subjected to the will of a person of the other sex, of being treated domineeringly by this person, humiliated and even mishandled. The idea is toned with pleasure; the person seized with it revels in fantasies in which he pictures to himself situations of this sort; he seeks to make them actual and not infrequently becomes more or less psychically impotent for response to the normal stimulus of the opposite sex through this perversion of his sexual instinct. This psychic impotence does not rest, then, on a *horror sexus alterius* (fear of the other sex), but only on the fact that for the perverse impulse satisfaction other than the normal, one through the woman but not through coitus, is adequate.

Thus, Krafft-Ebing saw in masochism a feminizing of the man, because love of service and of subjection is essentially feminine. Masochism to him was a pathologic overgrowth of specifically feminine elements, while sadism was looked on as a pathologic increase of the masculine sexual character.

In masochism, according to Krafft-Ebing, it is as though something belonging to the woman has passed over to the man as a perversion. The person of masochistic feeling seeks and finds fulfilment for his purpose in the fact that he ascribes to his partner male psychic sexual characters—here also in a perverse, exaggerated manner in so far as the sadistic woman represents his ideal.

Thus, Krafft-Ebing spoke of the (male) masochist as impotent and therefore obliged to deny himself possession of the woman. He insisted, however, on the normal attitude toward the woman and never failed to emphasize, even in the clinical histories, the fact that the patient was not homosexual. Masochism, according to him, is a reaction form which develops only on the basis of a psychopathic personality usually associated with a bad inheritance. The essential thing for him is the direction of the sexual impulse to the circle of ideas of subjection to the sexual partner and mistreatment by the latter.

Krafft-Ebing, however, appeared to overlook certain aspects of the situation. Sadomasochism is not merely a quantitative heightening of the normal sexual impulse, whereby sadism corresponds to the masculine, and masochism to the feminine, component of the sexual instinct. "Masculine" cannot be made entirely synonymous with "aggressive," or "femininity" with "submissiveness," for a sadistic disposition is found in women as well as men. It is well known that there are many weaklings among sadists, while masochists often are ideal whole men. Sadistic women, too, do not always correspond to the ideal of a powerful mistress and are often small and insignificant in figure, so that one would not suspect this degree of ferocity and lust for power.

Weaklings overcompensate in fantasy for their weakness in being unable to overcome another's opposition through an extreme fantasied sadism, in which the object is delivered over, incapable of resistance, into their power. Masochists, on the other hand, fear their own strength and escape from this fear into the passive rôle. The overcoming of the primary sadism expresses the greatness of the internal resistance.

There is recognized further as the important fact in masochism the avoidance of the normal sex act. The psychic impotence of the masochist no longer appears to be the consequence of masochism, but the

masochism the result of the impotence. The masochist seeks another object than the woman whom he has before him and with whom he shows himself to be impotent; the masochism serves merely to veil this secret sexual goal. Thus, one looks on sadism as a paraphiliac neurosis in which the will to power is sexually accentuated and masochism as one in which the will to submission is sexually accentuated. Here sexual pleasure is procured from nonsexual sources; stages preliminary to normal sexual relationship (partial fore-pleasures) have become aims in themselves.

Persons who express all their instincts are rarely cruel. The original cruelty in man appears in the normal person only when feelings of pain enter under the influence of unfulfilled aims. Cruelty and sadism, therefore, must be separated. Sadism is a paraphilia and means cruelty only toward those with whom one is emotionally involved. Ruthless actions performed toward persons concerning whom the feeling is neutral is not sadism. Sadism means cruelty toward persons whom one loves! Hate and love are bipolar forms of expression of one and the same feeling, with the distinction that love endeavors to subject the partner through pleasure and hatred through pain.

Nor is masochism to be confused with submission or obedience. If this were the case all submissive persons would be masochists. This is by no means true. Masochism is the surrender of a strong person through love. The sadistic roots of masochism can be repeatedly demonstrated. The masochist is the strong person who becomes weak through sense of guilt and through love. He may then, from fear of this weakness, fall into an attitude of defiance toward those he loves most. He does evil to them because he can offer no resistance to their love.

The phenomenon of "pleasure in pain" is observed among many masochists and finds expression in persons inflicting wounds on themselves or accusing themselves unwarrantedly of serious crimes in order to receive the punishment dictated by the unconscious. This comes from the sense of guilt that in itself is the result of sadism; this sense of guilt craves punishment on earth that it may escape the punishment of the hereafter. This urge to "suffer innocently" leads to all sorts of strange follies. There is the man who runs to the police and accuses himself of a murder which some one else has committed, and there are women who have a mania for operations, misleading experienced surgeons to attempt a variety of surgical interventions.

DEVELOPMENT OF SADOMASOCHISM; INFANTILISM

In opposition to those who view sadomasochism as a hereditary disposition that can only be evolved and not acquired, it can be shown, admitting the disposition to a strong impulsive life which all neurotic persons have in common, that it is a disorder due to the environment

and is more particularly traceable to definite influences in childhood; it is a form of psychosexual infantilism. The most transparent sadomasochistic type is infantile, a characteristic which it shares with other neuroses. A scene of childhood, sometimes so distorted through inversion as to be made unrecognizable, is repeated, in which the patient plays the child. Sadomasochism is thus a phenomenon of reversion. On this basis sadism is more strongly accentuated; it persists beyond the infantile period which is common to all persons. It is frequently turned into masochism as a result of the consciousness of guilt, remaining anchored to the unconscious. Because of the fact that the precipitating causes which go back to the earliest years of childhood have fallen under repression, the condition appears to be of a native disposition. The early history which the subjects give is deceptive and is designed to conceal the true origin of the neurotic structure. They like to give the impression that the condition is congenital with them.

Masochism universally develops on the basis of hypertrophic sadism. In childhood the patient may manifest every kind of sadistic impulse possible. He may torment animals and play with thoughts of murder, especially fantasies of poisoning. (It may be considered as talionic punishment for ideas of poisoning that some masochists have a pathologic fear of poison and attach a number of obsessive actions to it.) The patient is often pathologically jealous of one parent or of the siblings. He wants everything for himself—the love of his parents and even that of the servants. He wants everything for his own advantage and at his service. He is never able to grasp another's point of view, showing as well absence of ethical feeling toward others. These are all signs characteristic of the neurotic person, which he shares with the criminal.[3]

In the masochistic scene he holds fast to some memory and derives from it both pleasure and punishment at the same time—the familiar form of neurotic compromise.

With the development of the religious feeling and the sense of guilt, all the sadistic impulses and all the criminal thoughts seem to vanish. In this change from sadism to masochism the patient goes through a period of genuine suffering. But it is hardly to be expected that the fundamental sadism should entirely disappear. Rather should masochism be conceived merely as a painting over of the sadistic portrait. As one gets to the deeper layer of the mental content of masochists, sadistic elements appear more and more frequently and at the expense of the more masochistic elements. The dreams contain any number of sadistic

3. In this type one sees the neurotic person approaching closely the personality of the psychopath. I have long suspected that what is now regarded as constitutional psychopathy may, on more careful observation and study, eventually turn out to be but a particular form of sadomasochistic neurosis, with greater emphasis on sadism than masochism and with but little, if any, sense of guilt developed (Karpman, Ben: The Problem of Psychopathies, Psychiatric Quart. **3**:495, 1929).

scenes. It is significant that, in some cases, successful coitus is followed by strong feelings of remorse, which are entirely absent if the orgasm has been reached merely through flagellations. Analysis shows plainly that this is due to the presence of the dangerous incest fantasy, which is rejected by consciousness as unnatural and sinful whenever whipping permits a sense of reconciliation and reward.

The important factor that is never absent from the early history of a masochist is the significance of illness as a source of pleasure. Children fall ill and complain of pain, whereon the parents do their utmost to provide pleasure and minimize the pain. Unwittingly, an association is created in the mind of the child between pain and pleasure, and the inclination to masochism is fostered. On recovery from an illness, as normal attitudes return, the child cannot stand the sudden deprivation. This gives rise to outbreaks of anger and the inventing of all sorts of situations that will trouble the parents, which is at once a punishment and desire to recreate the original situation. A brother's or sister's illness may also be the starting point of a masochistic attitude—the desire to be ill, etc.

In the consideration of the organic background of sadism, Sadger laid great stress on the heightened eroticism of the muscles, skin and mucous membrane (compare susceptibilities of the skin to poison-ivy, etc.). Jones regarded increased anal and urethral eroticism as the prepared organic background. Havelock Ellis believed that the auto-erotic instinct of the child may display itself as the wish for flagellation without an act of flagellation having been witnessed or experienced. The flagellatory instinct, according to him, is often absent, but it is also present so often, apart from whether the child has had any actual experience of whipping, that he saw in it a manifestation of the auto-erotic impulse of childhood. His observation showed it to be more common in girls than in boys and more common in inverted men than in normal men.

SADOMASOCHISM AND RELIGION

One finds among masochists the tendency to degrade themselves and to apotheosize the sexual object and make possession of the latter more difficult; deeming such possession unnecessary, they avoid it. They find their pleasure in worship. Despite the representation of the bestial in the specific masochistic fantasy, the masochism is an ascetic tendency and makes its bearer psychically impotent, that is, actually chaste. It can be demonstrated that this ascetic tendency arises from religious motives and so must naturally lead to a merging of the ideal with divinity. Again and again one is able to point to the significance of the "prostitute complex" and its overcompensation. The prostitute is often made a saint.

Peculiar to sadomasochism is the strong intermingling of religion and eroticism, and the religious element is often strongly represented. Masochists conceal their religious nature only in rare and exceptional

cases. A certain amount of penitence and religious feeling is mixed in the paraphilia, which sometimes assumes a direct religious character (flagellation, asceticism, self-torture, withdrawal from every pleasure out of religious motives). The sadist's attitude toward religion is one of defiance. He is the blasphemer and atheist par excellence, while his inner religiousness manifests itself outwardly in a pathologic asceticism which renounces the highest goal, the possession of a woman.

A distinct religious expression of sadomasochism appears in a striking form in the lives of saints and martyrs who try to kill the lust of the flesh, as shown in the flagellants of the middle ages and in the manifold religious sects which inflict pain and injury on themselves out of love of God. These flagellants probably entertain prohibited sadistic trends and punish themselves for these as though they were accomplished facts. For in religious submission to the will of God the motives of humiliation, asceticism, penitence, self-torture and even self-mutilation and death are prominent. Such religious ecstasies appear at times to be considerably stronger than the sexual ecstasy, as the masochist would rarely dare permit himself such severe injury if at the appropriate moment he did not feel it as ecstasy.

The ascetic tendency of sadomasochism is unmistakable. In this respect the sadomasochist often resembles the fetishist. The significance of the religious sentiment in the dynamics of neuroses is in most cases inadequately evaluated. Masochism never arises without participation of the religious forces. The sadist becomes a masochist as the result of a deep religious sense of guilt, turning the original sadism against his own ego.

One finds in all these cases evident amalgamation of the specific scene with religious motives. Just as religion is permeated with sadistic motives (cannibalism, necrophilia, bloody sacrifices and the like), so the paraphilia is as often combined with religious tendencies. One need mention only the epidemics of religious flagellation, the self-scourgings of the saints, the tortures of the martyrs and the vast number of cruel ideas which are instilled into the minds of children in the way of religious instruction. It is unnecessary to waste words over this. A prophylaxis of religious sadism seems impossible with the fixed conservatism of the Church, and a change in the tendency to produce an effect through fear and horror is unlikely. The conception of hell and the idea of a mercilessly punishing Judge who lays a penalty for impulses which He himself has implanted reveal in themselves an almost sadistic character in the conception of religion.

RELATION TO OBSESSIONAL NEUROSES AND FETISHISM

One need not have the pessimism of Schopenhauer to appreciate the profound rôle that hate plays in life. It is not only much easier to hate than to love, to kill than to save, and to take than to give, but the reaction

is much more instinctive and effortless. One can hate in common much better than one can love in common. There is more than a streak of sadism in every human being; this element is a large component; indeed, much of social and personal life is organized on a sadistic basis, while culture represents the defensive barrier that society has erected against the onslaughts of sadism. In sadomasochism one sees the crystallization of this conflict in a relatively pure and concentrated form.

The investigation of the sadomasochistic paraphilia is like a journey through the inferno of human brutalities. The number of sadists is legion. A large proportion of them have sublimated their sadism and made it serviceable to culture; others have made it individually and socially innocuous through well functioning repression, and a very small number give expression to it in the form and manner here portrayed. They are all tormented by an evil conscience. They are unhappy because the polar tension between the ideal set by society and their ability to live up to it is so great. The neurotic person is a criminal without the courage to commit crime, constantly oscillating between good and evil. Considering the inner nature of man, it is surprising that the number of actual criminals is so small; no doubt this is the result of repression in the conflict going on between instinctive craving and cultural demands.

Sadomasochism is a complicated neurosis, approaching the obsessional in type. An obsession arises through repression of an idea unacceptable to consciousness and through transference (displacement) of the affect which has been set free to another apparently less painful, or even entirely innocuous, idea or action. Thus, a person having a feeling of dirt (guilt) may repress this feeling, be conscious only of being dirty and then show a mania (obsession) for washing in order to cleanse himself. This is essentially the case in sadomasochism. Most patients reveal obsessive symptoms; indeed the neurotic act as such impresses one as an obsession. The actual meaning of the neurosis appears to be hidden and is first brought to consciousness through psychoanalysis. The characteristic "death clause," which is absent from no obsession, does not appear openly in the sadomasochistic paraphilia; it is either masked as superstition or manifested as hate, which in the final issue is deadly. The sadist strives originally for the total annihilation of the object; every sadist is really a murderer. The attitude toward life is always pronouncedly bipolar. The splitting into two personalities which the obsessional neurosis so dramatically configures reveals itself also in these cases. The attitude toward parents and brothers and sisters is bipolar. This bipolar attitude leads to doubt and distrust. Nothing is certain; everything is vacillating; the paraphiliac situation seems to be the only stable pole amid fleeting manifestations.

The sadomasochistic behavior is only a pale symbol of the behavior whose place it takes. Being only a substitutive form of satisfaction, it can give but temporary and partial release. This effected, there soon

arises again the necessity for further activity, and the process is repeated endlessly because the real satisfaction is never achieved. The sadomasochistic neurosis thus shares with the obsessional neurosis the compulsion to repetition. It is a question of an undischarged affect, the abreaction of which is attempted in the specific scene of the neurosis (Freud). To the compulsion for repetition corresponds the narrowing of the field of vision. The sadist suffers from a fixed idea in Janet's sense. His entire affective life groups itself about this one idea or about the single scene which expresses this fixed idea. The fixed idea is anchored in the family, in which the specific scene is enacted, even if the substitute objects of the present appear to have no relation to the family. Frequently, the entire family is the secret harem of the sadist. In many cases the sadism may be traced back to the affective attitude toward a single member of the family. In consequence of this fixation on an infantile object, all sadists are incapable of love; that is, they are incapable of psychic love and in most cases are impotent. When there is potency the proper orgasm is lacking. The highest pleasure is always attained through an auto-erotic act, which means with the aid of the imagined specific scene. The fixation on an incestuous object is unconscious and is disguised through inversion and displacement.

The attitude toward the loved object is always bipolar; one current seems to be completely repressed. These patients usually love the object of childhood, while the hate component is split off and displaced on an object of the present to which they are indifferent, which is the obsession. This original bipolarity—that is, the coupling of love and hate—forms the basis of the sadomasochistic neurosis. The hatred directs itself partly from without and partly from within. It is not the entire personality that is hated, but a component of the triad, man, woman or child. The internal conflict is again projected externally and discharged in a symbol. The specific scene of the sadomasochist is to be understood merely as a figure or a fiction in which many infantile incidents are condensed to a single scene. This fiction is to be resolved like a dream or a cover memory, similar to the actions of the person with an obsessive neurosis.

A strong feeling of inferiority is characteristic of all these patients, arising from the endopsychic recognition of their asocial natures. Many patients have been trained to a sense of inferiority, while others have acquired it through social prejudices. The sadism of the cripple is well known. Here belongs also the sadism of illegitimate children and the sadism of the unfairly despised. Where society sows hatred it reaps paraphilia or criminality.

All these patients are incapable of love, yet seem consumed with the desire for it. They transfer this condition to the entire world about them. They crave recognition and sympathy, yet act as though they had no desire for them. They are all ambitious, yet too weak to carry out their plans for a great historical mission. Thus, envy and jealousy drive them

into the rôle of the person at once revengeful and penitent. They feel themselves cheated of their desired happiness and allay their pains in the pleasure of the wrong they can do themselves and others.

Intimately associated with sadomasochism is a well developed fetishism, in which one observes a still greater flight from the partner. Both types lead logically to obsessional neuroses, which represent the exquisite types of hate neurosis. To be sure, in the obsessional neuroses the original sadism and also its reaction formation—masochism—are so overgrown with neurotic symptoms that only through analysis can the action of hatred and conscience behind the strange manifestations be demonstrated. Being basically obsessional in nature, both types manifest themselves as repetition compulsions; through an obsessive symptom the affect is displaced, and the impulse is thus bound.

Therefore, one rarely finds an uncomplicated case of sadism or masochism. One must be prepared to discover various parts of the psychosexual infantilism, sometimes almost the entire program: homosexuality, anal and urinary sexuality, narcissism, zoophilia and so on.

Psychoanalytic experience shows that true cases of sadomasochism are like fetishism in that the end-result represents a flight from normal sexual relationship. In both types of neuroses one observes the masking of ascetic tendencies. If sexual intercourse takes place at the end of the sadomasochistic procedure it is an exception. There are intermediate and transitional stages, as is confirmed in fetishism, too. But in all cases a plainly ascetic tendency can be proved, which permits, as the only exception, intercourse with prostitutes, excluding respectable women from the system or reality, while phantasy plays freely about them.

RELATION TO HOMOSEXUALITY

In so far as masochists appear to be seeking the opposite sex they may be said to be heterosexual. But when one studies more closely the rôle they assume in the relationship, it develops that the relationship is reversed. It is the feminine in the man that seeks the masculine in the woman. The assumption that this is due to a partial feminization which has affected the secondary sexual characteristics finds support in the fact that heterosexual masochists reveal a feminine nature and actually present feminine features. Thus, it comes to be understood why masochistic traits are so frequently encountered in men with homosexual feelings.

Masochism appears as a substitute for direct homosexual expression. A number of neurotic reactions, especially those of many fetishists, could all be derived from the suppressed or open wish: I should like to be a woman. In the phenomenon of transvestitism, described by Hirschfeld, men feel happy when they can wear women's clothing and women the converse. Effeminacy in masochism may be so extreme that men may offer themselves to be castrated by women—the organic fulfilment

of a wish that "I might be a woman." In most cases sadomasochists are not aware of their homosexuality.

A man, in addressing his "very strict teacher and mistress," signed a feminine name. In these relations the patient played a sorry, pitiable rôle in a comedy of which he was author, chief actor and manager, while the "severe mistress" naturally only performed her part as her "client" wished to have it played. One sees here the masochistic abasement even in the assuming of a female name, by which the writer of the letter stripped off all manhood and took the humiliating position of woman, servant and slave. The rôle of mere servant did not satisfy him; he made himself a servant who, through neglect and disobedience toward the mistress, had sinned grievously. This wrongdoing deserved punishment, which, in view of the dreaded severity of the mistress, must naturally be severe. The penalty must consist of a brutal scourging of hundreds of blows, so that the blood runs.

On the physical side, one finds men with a small penis, incomplete descent of one testicle, an abnormally broad pelvis, flat iliac fossae, etc. Women may show marked tendencies toward the masculine, preferring to appear as men among men, smoking and drinking, etc. One notes here poorly developed mammae and large hands and feet—a physical accentuation of bisexuality—and a "phenomenon of reversion," as in all neuroses.

Strongly homosexual attitudes are observed in masochists in the withdrawal from the sexual partner. Men are impotent, while women with a pronounced masochistic attitude are anesthetic at coitus. It is true that the fulfilling of the masochistic fantasy brings with it orgasm. Yet the analysis of such fantasy shows again and again that it is the homosexual elements of the fantasy that release the orgasm. The attitude toward the opposite sex expresses itself in two forms: the *horror feminae* (fear of women) of the male masochists and the *horror viri* (fear of man) of the female masochist. In psychogenesis the two are quite alike.

Male masochists prefer women who have the secondary sexual characteristics less developed (if they reach copulation). Thus, lean women, those who have some growth of hair, and—a particularly surprising fact—elderly women, come chiefly into consideration. Indeed, a number of masochists prefer old, even gray-haired women to younger women. It is not only a matter of avoidance of the feminine type, the complete woman, but also of accentuation of the infantile, which has so extraordinarily great a significance in masochism.

The homosexual aspect of masochism reveals itself in the behavior and fantasies of the patients. Some of the patients are distinctly feminine with many passive traits. While showing great interest in eroticism and theoretical preoccupation with women, they evidence marked lack of skill in paying suit. They have a horror of coitus. Disquieting fears and repugnance often appear, even on the way to the brothel, and there are excitement, trembling, sweating and failure of erection and in all

attempts at coitus, for the paraphilia serves to discredit the woman for coitus through overvaluation and to conceal the homosexual disposition and the sexual aim. This disgust has its origin in repression. Such persons flee from heterosexuality for fear of committing lust murder. Besides repressing his homosexual component, the masochist represses also a sadistic attitude toward the woman, or he has an unconscious, incestuous sexual goal. The male sadist and female masochist are also homosexual and do not represent merely an exaggeration of normal traits, for the paraphilia is a matter not of excess but of inversion. The masochist hates women, often with a single exception. This hatred drives him to homosexuality. He therefore either becomes homosexual or flees into infancy: he regresses and becomes a child. Some masochists fantasy mutual masturbation with a boy and situations involving castration and cruelty: genitals being cut off, boys behaving brutally toward girls and girls being pulled apart. Some masturbate with the fantasy of being a woman or even a pregnant woman. On analysis even the fantasy of pregnancy turns out to be a matter of identification with some female member of the family. A female masochist will masturbate with the fantasy that she is the man in the imaginary act. The sight of a woman's body produces an orgasm in her.

The dreams are also suggestive of homosexuality: dreams of being a woman who is being taken possession of by a heavy man; dreams with a setting for heterosexual coitus, which is not realized; dreams of intercourse with a woman with a great deal of hair (masculine type), or of "beautiful" strong men having coitus with women; dreams of powerful masculine women, coarse-boned and having small breasts; dreams in which the woman is aggressive, and coitus inversus takes place; dreams of being a woman murderer and of cutting out the vagina from the living flesh and eating it, or of standing before the marriage altar and being married to a man.

AFFECT INTOXICATION; THE PROBLEM OF RESISTANCE

An insight into the origin of sadomasochism is obtained from a study of the behavior of children. Children do not well tolerate indifference on the part of parents; almost anything is better than that. Many are naughty in order to provoke some emotional reaction. Even blows and the anger of the person who administers them become evidence of love; it is an evidence of an interest taken in them. The affect of the one who strikes becomes the touchstone of his love, and even the pain is pleasure-toned, for it becomes a sign of the love. The overcoming of one's resistance is then felt as pleasurable and awakens the will to subjection.

The child is driven to the fixation of his primarily sadistic attitude if he finds that he is not loved enough, for his seeking for love remains unsuccessful. The first result is that he will compel the desired object

to love. The primary hatred is revived and becomes the reaction to his ineffectual wooing, the response to an erotic setback.

The problem of sadomasochism is a problem of affectivity. Excess of affect is able to transform pain into pleasure, as the apperception of pain becomes lost and is felt simply as excitement. This riot of affect arises from repression and from a sudden explosion of the choked masses of affect. In this affective debauch the specific scene may then receive from the polyphony of fantasies an excess of infantile forces through the appearance of the middle voices, without these middle voices coming into consciousness. During the specific scene the sadomasochist resembles a drunken person or a person under the influence of a narcotic. The affect serves the same purpose as the narcotic substance—it prevents the original affect from becoming clearly conscious.

In sadomasochism affect plays the chief rôle. The sadist brings himself to affect when he pictures the affect of his partner. In many instances it is not a matter of causing pain, but only of producing affects. The sadist revels in the fear, anger or humiliation of his victim; the masochist obtains satisfaction from the idea of feeling himself into humble and self-abasing situations. One strikes here the fundamental principle of the paraphilia discussed. For the sadist, it is a matter of overcoming the resistance of another; for the masochist, the conquering of his own resistances. The sadist dreams and desires situations in which a victim is bound and delivered to his pleasures. The bondage represents the impossibility of resistance. The lust murderer overcomes the resistance through slaying. Violence gets its character of lustful pleasure from the strongest resistance. The necrophiliac gloats over the defenselessness of the corpse. The masochist, on the other hand, craves situations in which he finds himself in bonds or in which he is forced or bidden, so that every resistance is impossible (command is the overcoming of inner resistance). The more humiliating the situation the greater is the pleasure. Analysis shows that this satisfaction is not produced without difficulty and struggle. The masochist reveals the most violent inner resistances; he fights against his paraphilia; he despises himself, but it affords him pleasure that he overcomes the inner resistance. At times, however, hatred against the one who has triumphed is awakened. Thus, many a masochist turns against his master or his mistress and slays him or her because of the original will to power; the sadistic attitude of hatred snaps back from its reversed form into the primary state, like the elastic band that has been stretched too far.

Psychoanalysis is always able to prove the origin of the sadomasochistic complex within the family. The resistance which the paraphiliac once experienced in the family is overcome in the present object of the sadistic fantasy with discharge of the components of hatred and revenge, while the masochist plays out his victory on his own body and psyche. Sometimes an infantile scene with reversed rôles or with reversed and

false affective tone is used. The inner play is then concerned with the overcoming of the infantile defiance, the breaking of the resistances.

It can be proved by countless observations that affective attitudes of hate toward the parents arise from the disturbance of an aggressive action or from the forbidding of onanism. Without exception, sado-masochists are onanists. Even when they have attained fulfilment of their paraphiliac fantasy, which is almost always a definite one, more or less narrowly circumscribed, they must masturbate with greater or lesser frequency. The onanism is, for the most part, an act of defiance. It is carried out despite the warning at the time and in spite of its being forbidden. The completion of the specific scene is impossible, since behind the fantasy images definite figures of childhood are hidden, and the sadomachochist knows that his condition has close relationship with incest. Onanism is the pleasurable gratification in which the external resistance seems to be totally excluded. Within there rages a stronger fight against the internal resistances, which later may be designated as phenomena of subsequent obedience, of the will to subjection to another's imperative.

All these patients are in a great degree incapable of love; the essence of their neurosis lies in the denial of the highest wishes to love and have normal relations. The ascetic tendency performs the function of a barrier, shutting out the end-pleasure as sin. It is evident that the actual inability for love in these patients corresponds to an inner prohibition. If they attempt to overcome this inner denial they strike on the inner resistance. Fundamentally considered, these paraphiliacs are impotent. They yearn for the highest pleasure. One knows of male sadists who, at coitus, despite a strong erection, can never reach an orgasm; of female masochists who avoid coitus and react with vaginismus or who remain anesthetic in coitus, and of male masochists who can never accomplish actual immissio penis. The enormous amount of affect under which they perform the normal acts discloses the strength of the inner resistance, which makes use of anxiety, disgust and shame to disarm the sexual instinct.

In analyzing the various cases, one always comes back to the realization that the chief thing in the fantasy is the overcoming of internal resistance. The sadist pictures to himself what is happening in the mind of his subject, whose resistance he calls forth and breaks. Only this feeling of himself into the affective life of the subject brings the expected pleasure. But the object is merely a reflection of his indifferent psychic and sexual components, and the scene represents a play with himself. A similar thing occurs in the masochist, who projects outwardly an inner constellation, undergoes a splitting of personality and is in a state to experience sadistic and masochistic feelings at the same time.

Thus, the decisive thing in the phenomenon of sadomasochism is not pain and the gratification derived from it, but the affect, which is of double origin. In the sadist, this arises from his sense of power in overcoming the resistance of another and from his feeling himself into the humiliation of his partner; in the masochist, from the overcoming of his own resistances (power over himself) and the feeling of himself into the partner who humbles him. These are not separate events, but polar expressions of a single complex.

The affect which is associated with the specific scene permits a transfer of the pain to pleasure. Hysterical anesthesias, heretofore regarded merely as conversion symptoms, should properly be considered from the standpoint of the displacement of affect. It is the affect which creates the neurotic anesthesia. There is a will to pain just as there is to freedom from pain. Some neurotic persons can voluntarily remove and produce anesthesia of the arms. Many heroic acts prove to be anesthesia produced by blocking of affect (Indian fakirs; the anesthetizing power of ecstasy in martyrs). Where a small stimulus brings a condition of excitement a stronger stimulus brings paralysis. Excessive pain causes painlessness, which explains the painlessness of some operations before the discovery of narcosis.

It is characteristic of sadists and masochists that they are sensitive to pain if it occurs without affect, that is, if it lies beyond the zone of their affective (sexual) life. Some masochists will submit to the greatest pain, but tremble before the dentist and cannot endure toothache. With the help of affect, pain is changed into pleasure.

All sadomasochists are affect-hungry, which is traceable to a definite repressed affect, a specific attitude of hate toward a person in the environment. Then there comes about a withdrawal of the hate from the original object and the turning of it against substitute objects or against one's own person. Human hatred, in the opinion of some, has its source in repressed love or pain. These overlook, however, the deeper truth or the reverse possibility that all delight in pain may be derived from a primary hatred.

INCEST AND JEALOUSY

It is not difficult to prove how easily a reversal of all sexual values may take place as a result of a too strong fixation on the family. The natural becomes sinful; kindness turns to hatred; the highest falls to the depths, and the lowest is exalted. As the need for love and hate vents itself on one's children, the new generation receives from the old the tendency to neurosis.

All sadomasochists are parent-sick. One seldom sees such refined cruelty as in sons who have wished to punish their fathers and daughters who have wished to punish their mothers for alleged want of love and understanding. As a rule, they are not aware that their entire conduct serves to cause their parents pain; they dissemble and pretend that it

was their duty to act thus; they themselves were unhappy that they could not do otherwise, and so on. It is seldom possible to convince the parents that this sadism is the result of a pathologic, all too great love. Next to parents, special attitudes are often established toward siblings, cousins and other members of the family. Repressed sadism may be derived from original jealousy toward a member of the family.

The relation to the incest complex is demonstrable in all cases. The paraphiliac man is obliged to renounce woman; the paraphiliac woman remains anesthetic. Both have set themselves an incestuous sexual goal, which is unattainable. The obstinate defiance allows them to act according to the formula: I will either attain the infantile goal or deny myself normal sexuality (the mother or no other woman; the father or no other man).

Of especial interest to criminologists are sudden expressions of hate. Sons often lay violent hands on their parents if the latter do not comply with their wishes. Sometimes such affective actions occur after a long period of harmony if some desire is not fulfilled, most frequently, if the parents, for important reasons, have opposed the son's marriage. One could readily cite a large number of cases. One case of this nature was reported in recent years in Vienna:

> An only son lived with his mother in the most harmonious relation. Then he fell in love with a girl of questionable reputation. The mother opposed the union. She sent her son to Italy for some months, and he returned cured of his love. They were talking once of this fancy, and the mother remarked: "Are you not grateful to me now that I released you from this unworthy attachment?" He made no answer, but as she went from the house to the garden he seized an ax and killed her.

Such cases reveal how strong may be the hatred which exists between those who are related, if unacknowledged incestuous tendencies disturb the friendly relationship. While otherwise the sadism seems to be excluded from the whole life, it is the particular delight of these persons to cause their parents pain and involve them in embarrassing situations, especially through the choice of unworthy love objects, certain that they will never obtain the parent's approval of them. The child begins to hate at the moment when some one places himself as an obstacle in his way. Children's outbreaks of hate manifest themselves in the first years of life as spells of convulsive crying (so-called holding of the breath) and later in attacks of rage or in epileptiform seizures. Jealousy is the well-spring of hatred and the most affective manifestation of sadomasochism. It is apparently an identification with another object and a desire for sole possession of this object. It gives rise to sadistic fantasies of revenge and the wish to put the rival or obtruding person out of the way. These are repressed and form the nucleus of the masochistic feeling of guilt and inferiority. The sadistic and masochistic fantasies are then variations of these original fantasies of revenge.

Jealousy derives its most important incentive from the anti-social side of narcissism, which is morbid self-love. It has been demonstrated that the masochist renounces possession of the woman and returns ever to his ego. His usually pleasurably toned activity remains the auto-erotic one, and the paraphilia is only an indirect course for the auto-erotism. Occupation with his own ego hides behind all these sadistic and masochistic scenes; the latter serve continually to represent the conflicts and division of the ego. The different pathologic variations of the sexual impulse are really reflections of the morbid condition within. Thus, the masochist, sadist and fetishist, while apparently seeking the object of their pleasure outside themselves, are in reality always engaged only with themselves. If they are jealous it is because their self-love has been wounded. They have so high an opinion of themselves that every love must only confirm for them their self-love. Every unfaithfulness leaves them in despair as to their own worth.

The sadist's hatred is first directed toward the family; then it leaps over on strangers, who are substitute images of the family. He becomes an asocial being, a recluse, an auto-erotist, a fetishist, a masochist, and comes to avoid everything that would lead him to be again a social person and to found a family.

To the neurotic person the family is but an extension of his ego. All sadomasochists and fetishists are family slaves and are never able to achieve release from the family. Each one wants to be a unique person, a case the like of which has never been before! Each wants to be the only one in the family, also, and has been the only one in the family either always or at certain definite periods (for example, during an illness). The primary attitude is hatred toward society (strangers and others) and love for the family (as the mirror of the ego). The more such persons wish to overcome the hatred toward society and transform themselves into social beings the greater is the resistance of the asocial forces that would rivet them to the family for all time. The hate must then direct itself against the family. These persons reverse their attitude, which changes to hatred toward the family and love for the group. They all make the vain attempt to conquer the love through hate, which, however, binds them ever more strongly to the family.

THE THEME OF THE DISSOLUTE MOTHER

The fact that the neurotic person often constructs a family romance in which he is not the son of his father and under certain circumstances not even the son of his mother (a changeling) reveals the effort to depreciate the parents and set a distance between them. The mother in this family romance is a prostitute. The image of the mother vacillates between the two extremes: goddess and prostitute. Again and again one is able to determine that in men the faithlessness of the mother and in women the Don Juan nature of the father are fundamental

sources of the sadomasochistic attitudes. The establishment of this connection is of deepest significance in the psychogenesis of the neurosis. The parents are despised and hated; this hatred is split off and carried over to the entire sex; the sense of guilt and the overcompensation result in a deification of the originally dishonored incest object.

The pull toward harlots arises from the fact that in some way or other the patients have come to consider the mother as frivolous and look on her as a prostitute; by her behavior the mother has killed something in them: faith in the purity of women. This appears to be more a matter of fantasy, although occasionally there is much basis for fact in it. Comparing these harlot fantasies which relate to the mother with reality, one finds that they are mostly invented combinations.

All the conflicts and bitter experiences through which the neurotic person has passed in childhood, the great disillusionment that the most sacred of all has become the basest and the fact that his belief in the infallibility of his parents has been destroyed—all these fantasies and facts which he has repressed—return again in his affective frenzy.

SYMBOLIC SADOMASOCHISTIC ACTS; CRIMINALITY

The impossibility of realizing their sexual fantasies compels neurotic persons to mask, conceal, reverse and symbollically distort their sexual guiding line. Behind this dramatic conduct the unfulfilled wish, which represents the strongest driving force in man's psychic life, hides itself. The sadomasochist always strives to portray the struggle of his impulses through a plastic symbolic picture in a specific scene. This scene becomes libidinized like everything else that has a sexual significance, whether the reaction to it is positive or negative. The neurotic sadist is forever in bitter warfare with his homosexuality; in the chastisement of his partner, he beats only himself. The man who is beaten may represent a woman, and conversely.

1. *Cannibalism, Vampirism and Necrophilia.*—Daily life abounds in sadistic reactions which usually escape notice; only the more gross brutalities come to attention. Cannibalism, vampirism and necrophilia are more extreme reactions. These are essentially sadistic, and, although humanity has long passed through the primitive stage that allowed these tendencies to come to open expression, traces of them are frequently found in the psychoanalysis of neuroses, and they are particularly undisguised in psychoses. One may look on them as atavistic echoes of a period in the development of mankind when such impulses were permitted frank expression. Fairy tales reveal many cannibalistic and necrophiliac scenes as residues of primitive occurrences—the saga of the vampire, for instance. Remnants of the custom of blood sacrifice are found in various religions.

In the main, these reactions appear as either symbolic acts or fantasies and are observed particularly in states preceding, accompanying or following an epileptic attack, but other neurotic persons (those showing simple absence, attacks of migraine or depression) also have to fight against these impulses, expressing them through fear and disgust as actions in forbidden fantasies. The preoccupation with the problem is evident in the peculiar behavior reactions of some people. Many persons become vegetarians who can eat no meat because it reminds them of "corpses"; some are unable to visit a cemetery without being overcome with nausea; others become sexually excited in cemeteries. Many peculiar sexual activities are influenced by the problem of death.

Cannibalistic, vampiristic and necrophiliac crimes are not so rare as is usually thought and almost always have to do with psychotic or epileptic persons whose illness was unrecognized. Necrophiliac acts may be committed under the influence of a superstition, as when the grave of a virgin is opened to cure some bodily anomaly. Murders have been committed to appropriate portions of the body, which were used or eaten for a particular purpose: to make a person rich, invisible, able to perform miracles, etc. The occupation in life may sometimes be influenced by necrophiliac and like impulses, e. g., gravedigging, the washing of bodies or the study of anatomy. The fantasies of many sadists are horrible, but usually an unbridgeable chasm yawns between fantasy and reality. According to Abraham, melancholiac patients frequently achieve regression to lower cannibalistic instincts, and their disturbances in eating are to be attributed to repressed cannibalism.

At the basis of melancholia lies the sadistic cannibalistic direction of the impulses. Hate always lurks in the depths of the psyche. Prominent in such persons is the capacity for incorporation or embodiment of the sexual object, even to complete identification with it.

Psychoanalysis of a melancholiac woman showed: (1) fantasies of castration and murder, which appeared as reactions to refused love; the desire for revenge, even beyond death; (2) fantasies of her own punishment and death (defense against sexual transgression); (3) fantasies of sex and power, of being ravished or abused sexually, and (4) cannibalistic and vampiristic fantasies: she thought of corpses of animals, of eating human flesh, biting her child, wanting to suck persons' brains, etc.

In another case of depression the first attack occurred following an accident when the mother was in danger of being run over by an automobile but was rescued. This made a fearful impression on the patient and she was depressed for a month. (She was unable to escape the deep consciousness of guilt and fell into a depression, a reaction to a death wish.) A second attack occurred after the mother's death and may in part be explained as the moral reaction to the death wish. Another determinant lay in relations with her brother. Her life plan was to share her brother's life. His marriage (the loss of a love object) contributed further to the depression. She had sacrificed her mother without having reached her goal.

2. *Symbolic Cruel Acts.*—There are many cruel symbolic acts which take the place of an intended murder. Some men are seized with an inexplicable fear when alone with a prostitute and are unable to perform intercourse unless another person is present (pluralism). They often carry concealed weapons (knives), which they rationalize as a protection in case of necessity. Some of these persons are lust murderers who fear that in the orgasm they may kill their partners and who, therefore, need the presence of others for their own and the woman's protection. Their dreams are filled with bloody scenes and murders: girls stabbed and lying in pools of blood, prostitutes murdered and the dreamer in court defending his innocence.

Some cases show a displacement of brutal acts on an animal. Thus, in one recorded case the patient would strangle a fowl before the eyes of a prostitute, following which he was able to perform coitus with great orgasm; without the bird, he was completely impotent. In this case the fowl played the rôle of the prostitute, whose neck he wished to wring. As a child, he had had great interest in the wringing of the neck. He would tear his sister's dolls to pieces at the neck. At the age of 4 years he threatened to wring the neck of his little sister, as he had once seen done to a chicken. At puberty he had observed all women's necks and wondered whether they could be broken. Later the strong infantile impressions were forgotten, and the fowl became the chief object, displacing the woman. He was thus no longer aware of his original tendencies to murder the woman.

Another neurotic patient pressed his partner's arm until she had to cry out. The arm symbolized for him the neck.

Some patients seize the throat and pretend in fun that they might or would like to strangle it. Others stab a sofa through many times with a knife, and this is followed by coitus.

Cases in which a slight insignificant action betrays the sadistic fantasy are numberless. There is the patient who must always hold in his hand a ball of newspaper when he performs coitus. He squeezes this ball hard before the orgasm. Another example is the patient who would like to choke his victim.

Love-bite belongs almost to the inventory of normal love. But one knows cases in which the bite has to be bloody if the orgasm is to be attained. Gnashing the teeth during coitus betrays the cannibalistic fantasy, which expresses itself also in the words: "I love you so much I could eat you up." The necrophiliac desires the absolute immobility of his partner. The woman must resemble a dead person, for only thus will the orgasm come.

More ideational sadism manifests itself in derision and humiliation of the woman embraced. The fantasy often reveals itself through an exclamation made during the orgasm, as when men during coitus use humiliating and insulting language. Some men experience the greatest orgasm when they have intercourse with their wives after a dispute which has led to action. Some can engage in coitus only if they maintain the fiction of overpowering a woman in clothing, throwing her to the floor or the sofa and holding her so that she is unable to move. In bed or after suitable preparations they are impotent. Only the resistance

of the woman makes them potent (probable mechanism of rape). Some add another requisite: the potency is conditioned by the absence of sexual activity in the woman.

3. *Symbolic Sympathetic Acts.*—Such positive human emotions as compassion, sympathy and pity often prove, on deeper analysis, to have a large sadistic component. Behind expressions of condolence and sympathy when one hears of the misfortune of another is the primitive reaction of malicious pleasure that such misfortune did not happen to oneself; it is in the nature of overcompensation. Unconsciously and involuntarily one compares one's position with that of the person with whom one sympathizes and is glad that one is not the person to be sympathized with or pitied, but rather the person to sympathize and pity. Sympathy in another's joy is much higher and appears more rarely than sympathy in sorrow. It is a part of civilization and culture that one changes the joy in another's injury to sympathy or pity, behind which cruelty may be hidden. Thus, pity is sometimes only repression and not sublimation.

A large sadistic component is found in the mechanism behind charity and benefaction. Within, these persons are gloating over the tortures of others, revealing the malicious pleasure and the primitive sadism that are behind it. To witness another's pain creates a certain satisfaction, which at times rises to a feeling of sexual pleasure. Pity may sometimes dominate one's entire emotional life. There are, as is well known, monomaniacs of pity. The objects of the compassion are varied and direct themselves according to secret unconscious attitudes. A pedophiliac, or a man who suffers from the fantasy "A child is being beaten" may found a society for the protection of mistreated children or join such a society. A gerontophiliac may find the lot of old people deserving of pity. An overvalued idea may find expression in pathologic sympathy.

Since true sympathy knows no bounds or exceptions and extends its feelings to every sorrow, the type of sympathy observed in many cases reveals an extraordinarily strong affect which makes one at once suspicious because of its narrowness. There are persons who exhibit a monomaniac compassion. They are sorry, it may be, only for ill-treated small children. They read in the paper of a child who is abused, and their tears flow copiously; their indignation knows no bounds. In the same paper is a notice of the ill-treatment and slaughter of adults in Hungary; that does not move them in the least. "It is not in my line," said an Englishman once. It does not fit in his system. He is interested solely in persons suffering from hunger, and the cry of "starvation" sets in motion his tear glands and his pocketbook. His interest, moreover, is in famine in an exotic, far country, while the need of his own workmen does not touch him. There are persons who will give millions to charity but will haggle with a minor employee begging for a slight increase in wages. There is the instance of a philanthropist who

explained to an employee that it would be impossible to advance him, that his business would be ruined, that the other employees would also want an increase, etc. He found a thousand excuses with which to quiet his conscience. But one could observe plainly at the same time the smile of malicious joy which betrayed the inner satisfaction of the strong man playing with the weak.

DREAM AND FANTASY LIFE

Even in dreams sadomasochists and the fetishists are not happy, for these are often accompanied by considerable anxiety. In many instances the dreams are only reproductions of the day fantasies. The dreams frequently revolve about the whipping of boys, about being beaten by men (the homosexual motive), about a woman with cane in her hand, about striking with the hand "the full buttocks of a portly, middle-aged woman" and about killing a woman (overcoming one's homosexuality). Along with this one finds dreams containing death motives, dreams of funerals and terrifying dreams of being buried alive. Erotic dreams occur mainly in a paraphiliac setting. Frank incest dreams are frequent.

DREAM 1.—The patient dreamed of lying in bed with his mother, kissing her feet while she played with his genitals. He was lying with his head toward the feet. He went from the foot higher and higher and kissed the knees. He wanted to kiss still higher and woke up with a pollution. This would appear to be a clearly incestuous dream: behind the woman in his masochistic fantasies was his mother.

DREAM 2.—The patient lay near a woman; she touched him, and he woke up with a pollution. He cannot remember the face of the woman.

Associations.—The patient never had dreams of actual coitus, but always these "touching" dreams. After the pollution he was tired and suggestible; the head felt foggy, and there was pain in the neck. The next day was a lost day; he was worn out, absent-minded and unable to work.

The physical after-effects of such a dream may be accounted for by the feelings of guilt because of incest which he probably committed in the dream. The psychically conditioned inner remorse and despair were displaced to somatic processes.

The fantasy life of these patients shows a prodigious development; not only their special sexual activity but also their personal and social behavior is predicated largely on fantasy life. Like all neurotic persons they are dreamers with their eyes open. Consciously, however, they are scarcely aware of it, and when they come for treatment they are able to relate only a small fragment of perhaps only one particular fantasy around which center all their onanistic activities. For purely pragmatic purposes these fantasies may be divided into two groups:

The Major Sadomasochistic Fantasies.—(*a*) "A child is being beaten." Paradoxical as it may seem, patients entertaining this fantasy had seldom been beaten in childhood or at all events were not brought up by the help of the rod. In school the sight of another child being beaten was never a source of enjoyment similar to that produced by the fantasy. The fantasy has feelings of pleasure

attached to it, as a condition of the fantasy the punishment must do the children no injury. At the climax of the imaginary situation there is almost universally onanistic gratification.

In the case of female patients (Freud) the fantasy discloses several stages in development: (1) "My father is beating the child," or "My father is beating the child whom I hate." The fantasy, then, is certainly not masochistic. It is more likely sadistic, since, although the child is not doing the beating himself, nevertheless he enjoys witnessing it (2) "I am being beaten by my father." This is of an unmistakable masochistic character. (3) Instead of one child, a number of boys (in the fantasies of a girl) are being beaten.

The origin is traced to the agitation of the child over its parental complex. The affection of the girl is fixed on the father; there is jealousy toward other children, and as being beaten signifies humiliation and deprivation of love, the idea that the rival is being beaten is agreeable. But with the process of repression the sense of guilt appears, and punishment now goes to the child producing the fantasy instead of to the other child. The patient has become masochistic largely from the sense of guilt; that is, masochism is not a primary manifestation but originates from sadism which has been turned around and directed against the self. To masochism belongs not only passivity but pain as well.

The beating fantasy is not a final one but only a cover memory in the sense of Freud. It plays the greatest rôle among all the sadistic and masochistic fantasies. There are a large number of masochists who have never practiced flagellation but who are aroused to the highest pitch of excitement through the idea that a child is being whipped.

The scene "a child is being beaten" is taken from a much larger fantasy picture; the beginning and end are wanting. The scene itself shows the widest variations. The person administering the punishment may be of either sex, of any age and excited or calm, and the blows may be given in hate or in love. The child beaten may be of either sex and may submit or struggle. The punishment may be just or unjust. Unjust punishment is especially pleasurable. There are usually onlookers, and the patient himself may be the spectator. He usually identifies himself with the persons in whose place he feels himself. The scene of forgiveness and reconciliation is of the utmost importance. The child has to beg for forgiveness and thank the person for the punishment, or he may refuse to ask pardon. The chastiser is tender toward the child or may go away indifferent. The great pleasure in most cases resides in the scene of forgiveness. The stern man or woman then becomes gentle. The contrast heightens the effect. These details must be carefully considered in each case. The child punished often carries his resentment for some time and has to be brought to a gentler mood. The form of beating also varies. In most cases it consists of blows on the nates. The trousers are stretched tight so that the form is visible, or the nates are bared, which increases the excitement. Sometimes the child defecates or involuntarily passes urine. Urination and defecation frequently accompany or follow the beating.

The scene is made use of by adults in order to produce the greatest excitement through the pumping of affect. A certain affective disposition permits or makes easy the entrance of fantasy. One always hears that the fantasies come when the patient is excited. Vexation and anger have a particularly stimulating effect. The vexation passes over the actual experience to the fantasy, but the specific scene is always an exercise of fantasy succeeded by another which hides within itself the triumph of the child beaten.

It is of importance that a scene of revenge follows that of the punishment, and this is most deeply repressed. The great affect in the beating does not arise merely from the oedipus situation, although it has relations with that. To give one

example: A child was beaten because he had disturbed his mother in a love situation. He was—which increases the humiliation—beaten by the lover ("Give the naughty boy a few good blows!"). The child had fantasies of revenge. After many years the fact of the mother's dishonor was repressed; the lover had vanished in the depths of the unconscious and the great fantasy of vengeance was at the same time lost to consciousness. There remained only the scene "a child is being beaten." This scene is a compulsive idea and conceals the most important thing.

Finally, beating corresponds to the need for contrast. The child cannot bear the lack of affect in its environment. The blows express the greatness of the affect and at once create a contact. Other important complexes, such as the castration complex, are also frequently hidden behind the scenes.

(*b*) "A woman is being carried." This fantasy appears with many variations. The masochistic man revels in the idea that he is compelled to carry a heavy woman until he almost breaks down under the burden. The sadistic woman may manifest the same wish as a token of her domination and the complete subjection of the man. More rarely one imagines that one is carrying a child or, in association with zoanthropic ideas, there is identification of oneself with a riding animal. The woman in riding clothes with a riding whip belongs to the last-named fantasy. Not unusually there is identification of a man with a dog which is harshly treated by a woman. One might trace this fantasy to the reversal of actual infantile situations. Children are carried around by adults, and exchange of rôles is a daily occurrence in the paraphilia.

The degradation of one's own person into the animal, the instinctive, follows as the bipolar contrast to the elevation and deification of the woman: On the one hand there are purity, absence of sexuality and divinity; on the other, uncleanliness and animal instinct. The polar tension between man and woman is increased and overstrained. It rests on the infantile prototype "mother and son," or "father and daughter." Reverence is mingled with love. The lower the masochist stands the greater becomes the feeling of the difference, which finds its sharpest expression in the picture "God and beast." This is the reason why the number of coprolagnists and urolagnists is so great among masochists.

(*c*) "A hand is being beaten." This fantasy is less frequent than others, but sometimes shows a remarkable condensation of symptoms.

II. *Minor Onanistic Fantasies.*[4]—(*a*) Purely Sadistic Fantasies: (1) A flagellation fantasy: The patient fantasied flagellation or chastisement of boys; these must be good-looking and of good families, and must wear sailor suits or linen knee breeches with navy stripes, and a cane must be used (probably a reproduction of an earlier scene).

In another fantasy the patient would like to undress his beloved completely, bind her naked with straps to the divan and flog her unmercifully so that he could enjoy her outcries and her helplessness. This wish appeared after separation. He wished to torture her as he had never done before, but only out of love. "I am a billionaire, have my own house with the cellar arranged for sadistic orgies. I abduct a lady in an automobile, bring her to me and compel her to go into the cellar rooms. The woman is seized there, strapped to a chair and whipped. She is angry at being thus taken unawares, but her cries of pain are of no avail." The fantasy was followed by an orgasm.

(2) A mourning fantasy: The patient had coitus in a death chamber. The woman was weeping over the loss of her husband. The patient rushed on her

4. Each fantasy is taken from the history of a different person.

and forced her to coitus. Despite her sorrow, he brought her to the orgasm. Sometimes sadism is a condition of potency (the patient may be roused chiefly by women in mourning, and satisfaction consists in overcoming their sorrow, or he may be able to have relations only if using insulting or threatening language).

(3) A torture-rape fantasy: A group of women, often mother and daughter, were first sexually excited and tortured at the same time. Torture with feathers was the ingenious form used. The women, stripped naked, were tickled with fine feathers until they were almost dead. They were bound and unable to defend themselves. A brutal negro raped them. The daughter was deflowered before her mother's eyes. The mother was at the same time violated in the presence of the daughter by vagina and by anus. The fantasies always ended with a scene of impalement; the moment the pale was driven in the orgasm occurred, together with the idea that the tortured woman fell in a swoon. (The many victims were an overdetermination of one woman, the mother.) The patient was an epileptic.

(4) A cannibalistic fantasy: "I am very rich and have a submarine. I carry off women to a lonely island in the South Seas. The fantasy seems too immoral; I change the woman whom I have carried away into a rather old, fat prostitute. I have hewn out of the rock on the island a place where I can take up my abode; there I flog the prostitute and afterward commit cannibalistic acts." Finally the prostitute was killed (choice of prostitute because this treatment does no harm with such a person).

(5) A castration fantasy: The patient imagined that he was a eunuch in a harem. A wonderfully beautiful girl was brought to an old and ugly sultan. The patient had to undress her and lead her to the latter. At the moment when the aged sultan forced the beautiful girl to his will the patient had an orgasm with onanism.

(6) A necrophiliac fantasy. The patient was roused to masturbation at the sight of feminine clothing. His onanism fantasy was as follows: "I imagined that I sat in a room where I had women in my power. After I had gratified myself by torturing them in every conceivable way, I imagined them to be dead and committed on their bodies every sort of desecration."

(7) A vampiristic fantasy: The patient asked herself anxiously what she might do if a young girl came into her power, docile and willing to submit. She said: "I should like most of all to kiss her breasts, then tear or bite them off and eat them. I would tear out the vagina, the uterus and the rectum. I would eat all of them and with them the inner portion of the thigh which borders on the sexual parts. I would then tear her open and "fondle" the viscera—take them out and put them back. I should like to feel their warmth. Finally, I should want to suck the blood from the side of her neck." The patient had often had the thought of killing a girl and doing all this to her, but she had never clearly considered the manner of death. During intercourse with a young girl she had repeatedly grown faint at the idea: "Tear out everything—rend it to bits."

She often had the fantasies: "I should like to get into the uterus or the rectum, lie there awhile and then break further into the abdominal cavity, where I could tread upon the viscera with my feet. I would at last find my way to the heart and there drink the heart's blood, then pluck out the heart and perhaps eat it" and "I should like to press into a man directly behind the testis. I would rage about his body until I came to the breast and there drink the fresh blood from his heart."

Her thoughts and waking dreams were occupied chiefly with the theme "blood." Her fantasies were "bloodthirsty" and made "the blood curdle." They reveled in cruelty and vengefulness toward a hated world. She spoke of blood as the symbol of love, hate, anger and passion.

She would like to suck and swallow blood but did not trust herself to do it, for she would not cause any one any pain. She could not drink red wine on account of its close relation to blood, but she was passionately fond of blood oranges.

She had thought of various things to do to the corpses of her husband and former lovers: chew the raw flesh but not swallow it, in order, as she said, to get the taste of the blood. She would like to suck the juices of the body, shell out or tear out the eyes and roll them around in her mouth so that she could feel their roundness and softness. Then she would fill the sockets with blood. She asked herself what color the eyes have after death.

She preferred sucking blood, which she might take from the hollow of the clavicle, to normal coitus. To her coitus and death were closely related.

She often had the idea of cutting off and burning up her husband's penis. Many times she loved her husband passionately: if he was submissive "like a child in my arms." But usually she wanted to be badly treated before sexual intercourse, and then she experienced great sexual excitement.

(8) A murder fantasy: The patient saw herself in her fantasies as an extremely beautiful woman surrounded by many lovers. She yielded to each lover for a certain period, then killed him. No trace of him was left. She destroyed twenty lovers thus. One of her fantasies was: A young and an old man came as suitors. She enticed the young man (her child's father) to murder the old man (her father). Later she killed the young man and lived in solitude forever in her beautiful palace.

(9) An incest-murder fantasy: The patient had fantasies of killing his brother-in-law and living alone with his sister, also of killing his mother and taking her place by the father. There was a frightful chaos of criminal ideas in his mind.

(b) Purely Masochistic Fantasies: (1) A queen-slave fantasy: In his fantasies the patient pictured a woman acquaintance as a queen who presided in a court of law with her subjects around her, who carried out the punishments. Because of treason against her majesty, the patient was sentenced to be beaten with rods and every possible scourging instrument. The final blow was always administered by the queen herself (overvaluation of woman).

During onanism he almost always had only masochistic fantasies; he was seldom able to picture coitus to himself. Sometimes the desire to be beaten by a woman and the longing for blows was so strong that he was compelled to masturbate. He experienced the greatest satisfaction in onanism when he pictured to himself his first girl acquaintance wearing red trousers with silk stockings when she must beat him.

(2) The patient's favorite fantasy was that he was a servant (pagism) of a personage in high position, for example, a count. The latter compelled him to have intercourse with a hateful female—a daughter, sister or wife of the count.

His anus fantasy was that he would meet a husky woman, and she would put him into her anus. (As a child, he believed that children were defecated from the anus.)

(3) A mistress-servant fantasy: The patient's entire effort was devoted to being subdued and humiliated by a "mistress." One of his fantasies was: "I shall be employed by her in debasing services, must wait on her when she gets up, at her bath and at urination. At the latter performance she occasionally uses my face and compels me to drink of the fluid."

(4) As a boy of 10, the patient had the fantasy that he was a lion and was beaten by a female trainer. (The lion is a symbol of the wild passions. This scene represented the punishment for his animal instincts and permitted the subduing of the animal thought to the woman in a symbolic picture.)

(*c*) Mixed Fantasies: (1) The patient fantasied women dancing dervish dances, who were flogged by an older woman after they had become exhausted. He imagined that he came into the scene dressed as a woman. (He had three ejaculations within one day). This fantasy shows the masochistic element, which is more visible because it is, in general, socially proper.

(2) The patient's ideal was a tall or stately woman inclined to Lesbian love and sadistic-flagellant procedure, who in his presence in the home circle would beat a pretty buxom girl of from 12 to 19 years of age. The desire was for a very large, comely woman, or, at least, one quite tall, intelligent and strong, with buttocks as large, white and well formed as possible, who would lay him between her thighs and strike him on the buttocks with a rattan or riding whip and who would at the same time subdue him psychically and bid him after the whipping perform cunnilingus or anilingus, in which he must obey unresistingly; otherwise he would be threatened with fresh blows. Coitus could be managed, but always with a certain indifference and perhaps fear of impotence, provided that the woman helped him over the technical difficulties by digital aid. The idea was rendered more pleasurable if he pictured a situation in which buxom young girls, young women, or pretty boys were chastized on the posteriors by an energetic, strapping woman with as large a rattan as possible. Such erotic relationship with an understanding woman was preferred to masturbation.

(3) The patient, who was apparently normal in every respect, was conscious of sadistic impulses. He masturbated occasionally, the masturbation fantasies beginning plainly as masochistic, but becoming later more and more evidently sadistic. In the beginning of the fantasy he was a slave of a beautiful young, powerful woman for whom he must perform most humiliating services which ended with cunnilingus. Imperceptibly the fantasy then changed. He was still her slave, but as her orgasm neared, he left off, and she became more and more the distressed one. As she moaned for gratification he ceased and let her implore, thinking up ever fresh tortures. Then she had to begin again, use every subtle effort to bring about an erection in him and to masturbate him, while he tormented her and left her unsatisfied. Not until she was half swooning for the experience did he thrust in the penis forcibly so that she lay as though dead.

(4) Following is a typical fantasy: The patient imagined that one of her former lovers was present and that she squeezed, pinched and struck him and thrust him violently with her feet. Finally she performed fellatio and anilingus. Then she desired him to tear the clothes from her body, beat her severely, throw her to the floor, fall on her with overwhelming force and finally accomplish cunnilingus.

(5) Although the patient recalled that as a small boy he was already masochistic, often losing himself in fantasies of being tortured, impaled and so on, further recollecting brought forth an earlier period marked with sadism. He had tortured animals in a variety of ways, reveled in fantasies of torturing persons and was particularly excited by the idea of being a cannibal and devouring human flesh.

(6) The first masochistic fantasies of another patient concerned boys as well as girls; the first sexual excitement (apart from auto-erotic stimulation) appeared in seeing boys beating one another when one boy sat like a rider on another.

(*d*) Paraphiliac Fantasies: The patient pictured to himself in succession all the women he knew and always from behind as they took off their drawers. He fantasied all sorts of occurrences: The woman lifted her foot high, or he performed coitus from behind. These fantasies concerned chiefly older women, once even his mother. Every one who had pleased him came into his thoughts. He carried out cunnilingus or anilingus or he had coitus and sucked milk from the

breasts. The favorite fantasy was that the woman was most flattering and caressing or that two men were having coitus with one woman.

SADOMASOCHISM AND EPILEPSY

Epilepsy, a Neurosis.—More recent investigations into the problem of epilepsy lay less stress on the symptoms than on the epileptic character which may exist without the seizures; but as yet there is no general agreement as to what constitutes the latter. However, as was long ago pointed out by Bleuler, epileptic persons are abnormal before the disease has put its stamp on them. Although there is no distinct epileptic character, yet the so-called epileptic person manifests certain characters of the neurotic person in a greater degree: he is distrustful, egoistic and sly and conceals his cunning behind simplicity, as he hides his pathologic sensitiveness and affectivity behind good nature and childishness. He is a strong hater, is envious and is liable at all times to transfer this hatred to the environment and the psychoanalyst. Trivial occasions often suffice to precipitate an attack.

According to Stekel, epilepsy is a neurosis, and the attack is an escape from pain into pleasure. Hate is the driving force in the epileptic psyche. The epileptic person retains his attitude of hate, which is primary to man, and it is through this hate that he falls into conflict with his environment, seeks protection in religion and finds it in his seizures. As a further expression of this attitude of hate, there is the strong reaction of jealousy of the epileptic person and the intimate connection between epilepsy and criminality.

The psychic component of epileptic seizures can be demonstrated with clearness in cases which appear to be precipitated by some acute situation. These "staged" attacks always appear if the epileptic person finds himself deprived of love, and his readiness for hatred would transform itself into the hate impulse. The mother, for example, wants to go to the theater. The patient is to remain alone at home. Before her departure there is a "colossal seizure," so that the mother has to stay home. Another patient progresses so far that he is able to take a position as stenographer. After a week he has an attack at business after his chief has reprimanded him. The epileptic person is able to react to every humiliation with a seizure, by which he revenges himself on his tormentor.

The Hate Reaction.—If the epileptic seizure is only a pathologic form of reaction of the brain, as Redlich believed, one still has to propound the questions how and when this pathologic reaction takes place. Experience shows that it is a matter of the affect of anger pressing toward action as an affective discharge of hatred, which is carried out in the uninhibited person and finds release for the impulse in the convulsion in the inhibited person. This anger manifests itself even in

early childhood; it takes the form of an affective convulsion, as all children show an increased readiness for convulsive seizures.

All investigators lay emphasis on the violent anger of epileptic persons, as well as on the apparent discrepancy between stimulus and reaction. The epileptic person is often given to terrific rages and affective explosions which are but equivalents of the seizures. He finds himself in a heightened affective readiness; the superficiality of the emotional reaction is a consequence of the narrowing of his mental horizon because he is under the domination of a fixed idea.

In psychoanalyzing many epileptic patients one comes again and again on their tremendous readiness to hate. In his inner nature the epileptic person is profoundly religious and has a heavy sense of guilt, which may be greatly augmented after the seizures. This inner piety leads him to suppress his hatred and often to overcompensate in love for his neighbor. He cannot endure the surging of his hatred. He flees from it into the unconscious. He often becomes outwardly gentle, kindly and helpful: Christ without and Satan within.

Fantasy Life.—The disposition to dissociation is responsible for the double life of the epileptic person: one of fantasy and one of reality. The fantasies find vent in sleep, in waking dreams and in seizures. Each attack entangles the epileptic more thoroughly in the web of his fixed ideas, brings him deeper into the realm of fantasy and makes him more and more introverted. The progressive character of epilepsy rests on the development of the introversion and is, unfortunately, often a result of wrong treatment.

These patients, since they live in a world of fantasy, escape every task which would divert them and utilize their seizures to make themselves socially useless so as to permit themselves to be maintained by their families or the state. They become more completely absorbed year by year in their dream world.

The Nature of the Attack.—Psychoanalysis in different cases of epilepsy has shown that every seizure is a matter of the acting out of an impulse. This impulse expresses itself in the various movements of the patient. The absence of the pupillary reflex corresponds to spasm of the pupils such as occurs up to a certain point with any strong affect or with every affective attitude. If one examines an angry child one finds transitorily some spasm of the dilatator pupillae. Janet referred to the fact that the concentric restriction of the field of vision is the somatization of the restriction of the mental field of vision. The epileptic in the attack is totally fixed on his one idea.

The attack performs the important function of defense. The deterioration and the progressiveness of the disease are not connected with congenital defect. They rest on the fact that the unconscious realm becomes all the while greater in extent, that fresh layers are ever being placed on the original nucleus, that new types of seizures develop and

that the structure of the latter becomes ever more complex. The splitting between consciousness and the unconscious grows greater, with the pleasure lying on the unconscious side both qualitatively and quantitatively. Finally, the pleasure principle overcomes the reality principle (Freud), and the patient tends toward the side of the greater pleasure.

Traces of different scenes, which are emotionally significant to the patient may be demonstrated in the attack. Such patients show remarkable resistance to treatment. One woman patient was once told by her physician: "You do not want to get well!" Later, she commented to her companion: "That wicked doctor! He wants to rob me of the most beautiful things I have ever found in my whole life! My attacks! I will not give them up!" This is indeed a will to sickness arising from the premium of pleasure which the sickness, in this case the seizure, affords.

Every narcotic measure enforces the state of day-dreaming and increases the introversion. The attacks sometimes cease with this therapy or become less frequent because the epileptic patient discharges his sadistic impulse in dreamy states or in profound slumber, for which he has no memory. The attacks which appear in many epileptic persons during normal sleep show that the epileptic patient needs a more profound narcosis than the usual slumber, the visions of which can be recalled as dreams if they are laden with strong affect. He requires total amnesia after the seizure. He dare not recall his fantasies.

Dostoievski spoke of this in the following significant passage:

> You healthy people have no idea of the feeling of joy that we epileptics experience before the seizure. Mohammed must certainly have had his vision of paradise in an epileptic convulsion, for he was ill of this disease just as much as I am.

He wrote once that he would give years of his life to be able to experience again this feeling of delight.

Regression.—The aura often begins first with feelings of sexual pleasure. Increase of the pleasure is experienced during the attack. One patient had as an aura a severe itching in the anus. He experienced in the seizure a pederastic scene with his brother, which had occurred some years before.

There is regression in all cases of epilepsy. The epileptic person becomes in the seizure once more a child and manifests the primitive original reaction. The discharge of feces and urine in the attack, eating and smearing with feces during the delirium, onanism and exhibitionism are to be considered regressive processes. They appear particularly in cases in which there is a uterine fantasy or a birth fantasy.

Sexuality.—Maeder is impressed with the connection between sexuality and epilepsy. Many epileptic patients masturbate in the attack or in the dazed condition which succeeds it. Maeder wrote:

> Masturbation is often found in connection with acute phenomena of epilepsy; it appears, for example, pre- or post-paroxysmally during a giddiness, in absences

and twilight states, but also in the intervals, especially under the influence of an affect, perhaps as an equivalent, if it is carried out in paroxysmal form.

Onanism is often shamelessly performed even by those who are not severely demented, particularly by younger patients (for instance, during medical examination).

Simulation.—The boundary line between simulated and genuine seizures can scarcely be drawn. The epileptic person prepares himself through pumping up his affect and brings himself into an affect intoxication which makes possible the precipitation of an attack. That a true Babinski sign can be simulated has been observed on many occasions. In studying many cases of epilepsy one becomes impressed with the readiness to convulsions, and the flight into the seizure can sometimes be produced with the aid of the will. The case of a man, aged 34, is instructive; he simulated attacks during the war in order to be sent home, and after the war was no longer in control of these seizures. He was then firmly convinced that he had challenged God and for this reason must be punished with epilepsy.

The Complexity of the Reaction.—One should not conceive of the attacks as formed on so simple a scheme. On one hand, every epileptic patient has various types of attack; on the other, the most varied motives are condensed in the seizure, as in the dream and, as Freud has shown, in the hysterical attack. The epileptic patient is at the same time active and passive; he experiences birth and death and sometimes rebirth. One thing is sure; the seizure imitates the joy in dying. It is well known that persons wish to die at the supreme moment of the orgasm. The life instinct and its polar opposite, the death instinct, find expression in the sharpest contrasts (Tristan, at the height of his joy: "Oh, let me die!"). Of course, it is difficult to bring the epileptic person to admit this death pleasure. The pleasure often resides entirely in the unconscious, in the fantasy that accompanies the seizure. What is visible after the convulsion is many times the blissful calm after the storm or often frightful wretchedness, as after a crime. The attack mimics the death struggle and dying (Kretschmer's death-posture reflex). The epileptic patient flees the dangers of life into a temporary death, then to be reborn.

It is difficult to comprehend the seizures in a system. One may, of course, consider the matter from the standpoint of the severity of the seizure and erect a scale ranging from absences to status epilepticus with its subsequent delirium. But if one is to take into account the psychogenesis of the reaction, a scale based on the strength of the regression is preferable. Such a classification is more desirable therapeutically, as the possibility of cure depends on how far the patient has regressed from the actual situation.

1. In his seizure the epileptic patient often passes through a definite recent experience. The movements and the facial expression imitate those called forth in a specific scene or scenes. A soldier who had strangled an opponent reexperienced the situation; he became cyanotic and his throat rattled like that of one who is being strangled. The petechiae were a result of the hyperpnea, which is attributable to the idea of strangling. A girl whose father came home intoxicated, tore off her clothes and deflowered her reexperienced the attack in her seizures, even repeating the cry which she uttered. This type resembles most the hysterical and has been called hystero-epilepsy.

Thus, a seizure may mean reproach, pleasure, etc., as the case may be. Sometimes a part of the body (arm, shoulder, etc.) that has been particularly involved may become completely anesthetic. Sometimes the same patient may have different types of attacks because of different motives; again, one attack may show a condensation of motives.

In general, it can be shown that seizures are not chance reactions, but occur in response to definite situations, however concealed these may be. Thus, one patient saw his mother attempting to commit suicide. He procrastinated in going to her aid and by the time he came she was dead. Universally he now has seizures whenever the associations are connected with the dead, cemeteries, hanging or choking. In his attack he drops his head forward in the position in which he saw that of his mother hanging and sticks out his tongue. According to Tremmer, epileptic patients are frequent visitors to cemeteries. In the psychoanalysis of epileptic patients necrophiliac tendencies frequently come to the surface.

2. Many epileptic patients enact a scene of strangulation. This fact explains the phenomenon of Tsiminaki, which is the precipitation of an epileptic convulsion through compression of the carotid. But closer study reveals that the phenomenon has nothing to do with the carotid, for it also appears if one takes the patient by the throat and lightly chokes him. It is the choking fantasy which releases the seizure. As the patient is seized by the neck he has an impulse to choke the opponent; this sets free his choking fantasy, which is in a state of affective preparedness.

3. Sometimes the epileptic seizure expresses a trauma of early childhood (regression). Maeder reported the case of a woman who had rare seizures. From partial psychoanalysis it was learned that she had experienced a sexual attack when she was 5 years old and again at the age of 12. The seizures began shortly after the second attack. A long dreamlike state followed the seizure. In this long dream (twilight state) she had sexual intercourse with her former instructors, the pastor, the factory director and overseer and certain physicians who had treated her. This case shows regression to the fifth year of age.

4. Strange as it may seem, the seizure may repeat not only the scene of birth but even of the intra-uterine period. There are persons in whom desire and ability are in glaring contrast, and reality and fantasy do not meet. Their thoughts are always wandering to childhood. Such a patient is completely dominated by the fantasy of the mother's womb. He sometimes lives in solitude so that he can give himself up to his fantasies and be the "only one." Sometimes he comes from a large family of children. He has the grotesque fantasy of killing his brothers and sisters in the mother's body and of coming into the world as the first-born. This type is extraordinarily frequent. One seldom finds an epileptic patient who has not passed through rebirth in one of his seizures. This has been confirmed through the careful investigations of Pierce Clark. These cases present the character of progressiveness (deterioration), and ultimately the patient becomes absorbed in his embryonic existence—he becomes demented.

The fantasy of the mother's body is frequently associated with the castration complex and a fellatio fantasy. The father thrusts his penis into the patient's mouth in the mother's womb; the penis is then bitten off. The frequent tongue-biting of the epileptic may sometimes be traced back to this fantasy. This active castration impulse is changed through the evil conscience (talion) into a pathologic castration fear.

5. In the seizure, the epileptic sometimes experiences his own death. In the case of the man who experienced his mother's hanging, which has already been mentioned, the identification is striking. This type corresponds in part to those mechanisms which Kretschmer called the death-posture reflex. There are persons who occupy themselves much with the theme of death. Suicide is frequently a talion for the wish to kill another or to see him dead. (The person who hangs himself in his seizure also belongs in this category.) Psychoanalysis then becomes more complicated because the epileptic patient acts two parts: that of being killed and that of dying. He is the other person and also the ego. (One sees continually that the categories merge into one another and that this presentation of the theme is not to be taken didactically.) Connected with dying are often visions of the last judgment, paradise and hell, which appear clearly in postepileptic delirium.

6. The seizure may express the committing of a forbidden sexual act. A large number of hysterical seizures belong here. The coitus fantasy is a forbidden act to chaste maidens. Normal coitus may be experienced in the convulsion. Much more frequent, however, are paraphiliac acts: cunnilingus; homosexual scenes, especially incest; active and passive scenes of rape; orgies; zoophiliac scenes, and the like. Many of these fantasies are sadistically colored, so that through them one arrives at the following category, which may be considered the most important.

7. The epileptic commits a crime in his seizure. The greatest regression takes place in these cases. The patient sinks to the level of "primitive man." One may assume that this regression goes back many thousands of years and removes all the inhibitions of civilization. The most extreme sadistic paraphilias belong to this category: vampirism, necrophilia, cannibalism, lust murder, and the sadistic frenzy for blood that manifests itself in mass murders. These sanguinary tendencies are frequently clearly expressed by the patient's actions. Tongue-biting is hardly accidental, but is an expression of the desire to bite and suck blood. The convulsive clenching of the fists often expresses the impulse to strangle.

In the light of this, the epileptic symptom complex must be considered solely as a self-protective mechanism. The patient collapses in order not to have to commit a crime. The self-defense does not function in some cases; the affective frenzy sets in before the moral ego can bring about the fall, and murder is committed.

Epileptic crimes are characterized by special brutality.

> A man, aged 38, gentle, kind-hearted and charming in disposition, manifested a deep love toward his wife. One day, without any particular occasion, he killed her in the most horrible manner, throwing himself on his victim. He smashed her skull and ribs and cooked her liver and other viscera. After the deed had been committed he went to bed and slept profoundly and quietly. All memory of the crime was gone when he awoke (Knowalewsky).

One sees here plainly the outbreak of cannibalism, which can so often be demonstrated in the fantasies and dreams of epileptic patients. Fortunately, such deeds are rare, and the moral ego sets itself energetically in defense against the "sadistic id." An epileptic peasant who had horrible impulses to commit murder during his seizures was aware of their approach and begged to be bound before they occurred. "When they seize me, I must kill, choke, even if I see a child before me. . . . Save yourself, mother, or I shall have to strangle you!"

One is reminded of the frequent cases of compulsion neurosis in which murderous impulses appear in the open. The patients consult the physician because they are afraid that they may commit a crime. One sees men who have murderous impulses toward their wives, mothers who feel them toward their children and sons who feel them toward their parents, all of whom wish to insure themselves in every possible way against carrying out these criminal tendencies. In no instance, however, does one see such a neurosis pass over into the epileptic symptom complex. The obsessional neurosis seems to afford sufficient security against the crime.

The mother who is afraid of stabbing her child will have no knife or other sharp-pointed object in the house; she will not stay alone with her child, and so on. It is different, however, with the epileptic person. He saves himself from crime like the person with an obsessional neurosis,

with this difference: in the person with an obsessional neurosis the criminal tendency is expressed in a day-dream, in the epileptic patient in a seizure. Persons with a compulsive neurosis rarely dream of their impulses. In the dreams of the epileptic person, however, one finds countless allusions to his desire and sometimes also the representation of his specific fantasy.

The material just cited suggests that epilepsy is a form of neurosis and its treatment should be psychically approached. While the epileptic symptom complex may be organically determined, forms largely psychogenic in nature are frequently observed, too; a psychogenic superstructure is often formed on an organic basis. There is no symptom of idiopathic epilepsy that may not with equal frequency be found in the purely psychogenic form. Psychotherapy may justly claim that in some cases the condition can be cured, in some improved and in some at least arrested.

SADOMASOCHISM AND SURGERY

The study of the life histories of sadomasochists and fetishists clearly points to the importance of psychic factors as molding influences. Granting fully the presence of constitutional factors in these cases, for many years and perhaps even for generations to come psychic phenomena will have to be explained largely in terms of psychic causation; since there is little if anything that one can do about changing one's heredity, the treatment must be approached through psychic methods. Only psychoanalysis can penetrate the family history. One who is content to listen to the patient's accounts, which emphasize and make much of those sadomasochistic features in his childhood which are common to all children, will never grasp these conditions and will insist on organic causes. The patient then becomes the victim of his physician. There are, unfortunately, many physicians who imagine that they can deliver a sadist from his illness through some operation. Patients have been castrated in the hope that their fantasies would disappear, but after the operation they continued to indulge in endless day-dreaming. The species of paraphiliacs who long to be castrated represent an attempt at feminization by means of castration.

The masochistic component is strong in many clinical pictures. Pain frequently serves to hide a secret pleasure and gives an opportunity for indulgence in a definite fantasy. Every year sees a number of patients on whom the most unbelievable operations have been undertaken, while psychoanalysis is able to determine the presence of a neurosis in the center of which is the pleasure in pain. The pain conceals a secret delight and at the same time represents the voice of conscience (self-punishment!). Many patients with severe neuroses have been operated on without judgment and without result.

FETISHISM

Relation to Sadism.—Mention has already been made of the intimate relationship that exists between sadomasochism and fetishism. One may think of both as having a common origin (which is sadomasochism) and proceeding together until they come to a fork in the road, from which one branch continues as sadomasochism and the other proceeds as fetishism. While it is true that fetishism may exist without sadomasochistic fantasies, sadomasochism is the root of fetishism, because it keeps the patient in a constrained compulsive mood and is therefore masochistic (thus accounting for the martyrdom). Common to both are:

The predominance of fantasy life over reality.

The more or less complete depreciation of the sexual partner and a tendency to flee from him or her.

Onanism, which is universal and is accompanied by fantasies. (The habit of masturbating before a mirror is characteristic, an expression of narcissistic love present in every case of fetishism.)

They affect intoxication in the respective scenes which are symbolic expressions of the conflicts.

Incestuous attachment, which is universally present, the incestuous love being repressed hate subsequently directed against the whole sex. Indeed, the incest wish is one of the never-failing bases of the paraphilia. These wishes cannot be attained in reality and are, therefore, transferred to the realm of fantasy, while in reality the trend is symbolized as a preference for some article of clothing or some other object. The formula followed by the fetishist is: "If I cannot attain the desired object [mother, sister, father, brother or some surrogate] I shall reject any other partner."

The relation to homosexuality and the obsessional neuroses.

The originally sadistic attitude, with feelings of guilt and penance as subsequent attempts at correction.

As in the sadomasochistic scene, there is an almost unbelievable condensation in the fetishistic symbol, the symbol (the fetish) being the focus of all the rays of the subject's phychic life.

It remains now to outline briefly the points at which these two paraphilias diverge.

Erotic Symbolization: In fetishism, more perhaps than in any other form of neurosis, symbolization plays a significant rôle, and the reaction, therefore, cannot be understood without a proper appreciation of the meaning of symbolization. Rank and Sachs defined the symbol as a "special kind of indirect expression distinctly different, however, from related forms such as the simile, the metaphor, the allegory and other types of pictorial expression or thought." White and Jelliffe called the symbol an "energy carrier" and this is certainly true, for the symbol not only contains the idea which it is supposed to represent, but it may have a plurality of meanings as well. In a sense, symbolism is primitive, like an ancient tongue which has almost died out, leaving stray remnants of its former fulness here and there (Freud). It is the language of a lost period of our lives (Jung). The concept of the symbol is indefi-

nite and often undergoes remarkable changes. It may change in a degrading downward direction or may come to have finer philosophic and cultural meanings.

The symbol of the normal person and that of the neurotic person represent entirely different concepts. The neurotic person exploits the symbols of the normal, but fills them with new meanings. Erotic symbolism and fetishism pay no attention to the general significance of their objects or to their usual symbolic value.

Fetishism is a paraphilia in which the sexual partner is dispensed with and the subject gratifies himself onanistically with the aid of a symbol. The symbol may be any object used by women (blouse, petticoat, chemise, handkerchiefs, apron, underwear, silk stockings, etc.), as well as (tight fitting) trousers, (tight) shoes, heels, bandages, suspenders or any object used by the person: neutral objects, such as keys and shoe nails, and even sausages. There is not a single article of clothing or an object of daily use which may not become an object of fetishism. Even parts and organs of the human body, such as pubic, axillary and head hair, tresses, pigtails, queues, finger-nails and toe-nails and the muscles of the calf (rubbing against which alone induces orgasm), may be such symbols. The latter group forms a transition to partialism, which is defined as the special attraction exercised by certain parts of the body or by certain types of persons in preference to other parts of the body or other types of persons (tall or short, slender or stout, blond or brunette persons, persons with a certain color of the hair, eyes, etc.). There is also partialism for wooden legs, bearded women, pregnant women, persons on crutches (masturbation may accompany fantasies of women who hobble on crutches), those with amputated legs, cripples who limp, etc. These cases have a sadistic basis, and the cruelty of the weak and that of the stronger toward the weaker play an important rôle.

Erotic symbolism has the tendency to free the subject from the necessity of a sexual partner, the fetishist in this respect succeeding more than the sadomasochist. The genuine fetishist becomes progressively freer and gradually retreats into an asexual ascetic, homosexual or autoerotic life. When avoidance of the partner is not complete and heterosexual attempts are made, there is either failure or no gratification.

As an illustration of erotic symbolism, one may cite the instances of fetishists who admire tightly fitting shoes, whether worn by a man or a woman, and of those who admire red, swollen, inflamed, dirty, sweaty feet. The sweat may represent blood, the patient fantasying a nail piercing the foot (Christ neurosis); or he fantasies that he is the one having sweaty, swollen feet, which fantasy would induce orgasm on masturbation. The sexual symbolization of the foot and shoe is old as well as universal. The female foot is the symbol of fruitfulness because it touches the earth; in places the shoe is regarded as a symbol

of the vulva, while the foot is a symbol of the penis. The perversion may be displaced from the foot to the neck, and compression of the throat becomes as gratifying as compression of the phallus.

Negative fetishism is only a special variety of fetishism. Some may love women who wear pince-nez, etc., while others hate pince-nez, mother-of-pearl buttons or certain types or all kinds of underwear. The buttons may be symbolic of nipples, which further stand for some female member of the family who is a rival for the mother's love. The fetish in hate is ultimately shown to be a love fetish.

The Meaning of Fetishism.—Fetishism, especially, performs the important function of insuring chastity to the fetish lovers. Paraphilia, in general, keeps the patient from the opposite sex. These persons are all infantile; to them coitus, *immissio penis in vaginam,* is a cardinal sin, while pleasure derived from other erogenous or sensitized zones is not regarded as sinful. They are ascetics struggling for purity and chastity and also betray a secret wish: the divine transformation of the carnal spirits. Fetishism is a religion of hate, which, though well disguised, breaks forth on occasions (in dreams, fantasies, etc.). It is a compulsion which the patient forces on himself out of motives of atonement. The fetish must be compressive and discomforting and must keep the patient under constant tension; that is why shoes, gloves or corsets are so often chosen. The thought of pressure produces an erection. But fetishism is not only a second religion; it is also a disease, a parasite incapacitating its host for any other form of mental endeavor.

There is no fetishism without a sense of guilt. Fetishism is a self-inflicted punishment for the patient's cruel fantasies and attitudes. In order for the fetishist to fulfil his reaction he must have compulsion, pain or coercion. Like all neurotic persons, the fetishist is a criminal without the courage of his impulses. In atonement for sadistic fantasies the patient constructs the sexual idea of asceticism, converting sadism into masochism. He is masochistic, tortures himself, denies himself the joys of life, is tormented by compulsions and has guilt conflicts. The fetishist executes tortures on himself and in a more specific way than the sadomasochist, binding himself with corsets, assuming the cruciform position, etc. (Every true fetishist suffers from the Christ neurosis—the chastity complex.) Symbolically, the female is a sign of chastity, whereas in life she is the incarnation of sin, because fetishism develops the depreciation of the woman. The pain experienced in tight lacing (of corsets or shoes) expresses the sadomasochistic component.

Universally there is a marked lack of conscious appreciation of the motives back of the reaction. The neurotic person does not understand himself because of the splitting of the personality, expressed in the bitter struggle between the instinctive and the moral ego. Fetishism, like all neuroses, is a curious combination of synthesis and antithesis, a compromise representing an ineffectual synthesis of both tendencies.

The fetishist stands between two powers, his soul torn between moral and amoral imperatives, and finally arrives at the impossible compromise of satisfying two antithetic demands at once. With the fetish as his God (just as it is to the primitive man), he founds for himself a new religion. But the fetish is only a caricature of a God. Thus one arrives at a formula which is almost paradoxical: a combination of divine ideal and animal craving, of God and Satan, both in one.

Fantasies.—Onanistic fantasies are frequently sadistic. A male patient fantasied two women struggling, one overpowering the other; a female patient fantasied killing new-born infants. Others fantasied whipping scenes and so on. One patient fantasied that he had a castle, had many girls serving him and later would cut off their hair.

The Harem Cult.—Characteristic of the fetishist is the harem cult—the collecting from women of articles belonging to them, attaching different values to these and changing the favorites like the pasha in a harem. He also has his Bible, a book (album) in which is a collection of articles belonging to women, perhaps of those with whom he has cohabited. The volume may also contain a series of obscene pictures of genitals and extravagant breasts and notations. One also finds among these patients the totem cult—fear of the deceased.

Kleptomania.—Associated with fetishism are marked kleptomaniac tendencies, as one might expect, since it may be difficult for the fetishist to obtain his symbol through normal channels. These impulsive acts are, as a rule, not recognized and pass as common thefts. The patients are often found roaming in a dazed state at the time they are about to commit the impulsive acts or after having committed them.

PROPHYLAXIS

The prophylaxis of sadomasochism and fetishism must be directed to two sources, the family and the school. Punishment only tends to heighten the child's sadistic reaction. Servants and educators frequently take vengeance on the children for the oppression to which they have to submit from the children's parents. Teachers are also often embittered and dissatisfied with their lot in society because they are so poorly paid and express their bitterness on the children entrusted to their care. Every position of power is used by weak persons to gratify an intensive will to power, if this will to power is sexually toned.

The mistreatment of children forms the saddest chapter in the history of mankind. Especially reprehensible are those forms of abuse which are draped under the ethical mantle of "pedagogy." It is certain that only love can educate. One should always endeavor to avoid punishment, whenever possible, and punishment need never be corporal. Forbidden participation in certain favorite games, perhaps exclusion from the classes which the child likes and which interest him, would suffice.

The ideal procedure would consist in convincing the child that his conduct is antisocial and in entrusting to him the right of self-punishment. Repentance and frank confession of the wrong should be considered adequate penalty. This slight punishment should be carried out only in case of relapse.

Parents and educators who beat the children entrusted to them commit a crime toward the children and toward the state, because such treatment unfits the children for life. Only those freely trained are actually free. There is urgent need of free men and women. Few parents and educators will admit that they experience pleasure in flogging. One often hears the untrue statement: "The whipping hurt me more than it did you!" These words merely reveal the masochistic feeling of oneself into the place of the victim and are to be considered as the expression of the pleasure in pain.

The hatred of the parents sows and reaps the hatred of the children. The number of cases of ill-treatment of children which comes to the knowledge of the public seems to be increasing. The child is felt as a fetter in unhappy marriages. Parents who were themselves sternly treated and were beaten are likely to continue this system of education. (This is the curse of the evil deed, that it is constrained to go on bearing evil.) Sometimes the child serves to recall a wrongdoing. The mother knows that it had another father; the father suspects this, and the child is made to feel it. Children born before marriage are often the object of particular hatred. They have forced the father into marriage and remind the mother of her "wrong."

Many of the patients are products of unhappy marriages. The unfortunate marriage conceals a twofold danger. On the one hand, the parents, because of their own disappointment in love, may transfer on the child their entire affection. On the other hand, the hatred may be projected on the child. The children are the bond which makes it impossible to dissolve the marriage. The unconscious hatred works then as a motor, discharging itself in a tendency to punish and in severity in training.

The establishing of the fact that a child may come to grief because of the unfaithfulness of his parents, that his life is shattered because the mother has an extramarital relationship, points one way to the prophylaxis of these neuroses. Unfortunately, parents have no idea how early a child receives sexual impressions. The sexual life of the parents is their private affair. One knows well that mothers who stand high ethically may yield to a lover under the pressure of their sexual need. But it is the parents' duty to guard their children from all impressions which might poison their fantasies. Physicians should work toward enlightenment in this respect and concern themselves with the mental hygiene of the nursery. The intercourse of the parents which is overheard (paraphiliac listening), scenes of strife and reproaches for unfaithfulness

made through jealousy may lay the foundation of a sadomasochistic and fetishistic neurosis.

Children's hate often springs from jealousy, and one cannot overestimate the overwhelming rôle that jealousy plays in the psychogenesis of sadomasochism and fetishism. Jealousy develops in persons who have not found satisfaction in love. Hence, the best prophylaxis for these paraphilias lies in education. What society needs is a campaign against envy and jealousy. Everything should be excluded from the family circle that could justifiably excite jealousy. Weak and sickly children are often coddled. This leads to jealousy of the healthy child toward the sick one, and also sets an example of the advantage to be obtained from illness. The mistake of partiality to certain children, overindulgence, and holding children up as models to others, is illustrated often in case histories. The love of parents and teachers must be impartially distributed. Envy and jealousy should be combated early. Children's reactions of defiance are usually the result of jealousy. If the parents are psychologically trained, they will recognize the jealousy and make the child conscious of it, so that he may overcome it. One has likewise to reckon with the phenomenon of unconscious jealousy, which leads to the most dangerous reaction formations.

Children's hatred has still other sources. Children will not tolerate the parents' interference in their sexual life. The forbidding of onanism and the punishment of infantile sexual play readily produce an attitude of hatred in the child toward the parents and toward all society. The child's right to his sexuality, which is now recognized as always present and necessary to his development, is not yet well established. Parents and educators would do well not to trouble themselves over the sexual life of the child if it does not exceed certain boundaries, that is, if it does not transgress the canon of the normal.

These are not rigid maxims or set rules, but merely suggestions derived from studies of cases. As yet too little is known of the inner psychic life of even normal children. Intimate life studies are great time-consuming efforts, but they pay rich dividends in the understanding of the growth of the human personality. In a larger consummation of such studies lies the only hope for relieving the suffering in nervous and mental diseases.

CRITICAL COMMENT

Although the literature on sadomasochism and fetishism is extensive (Krafft-Ebing, Forel, Hirschfeld, Bloch and others), nothing was known of their underlying mechanisms and therefore of their meaning until the advent of psychoanalysis. One can go through the contributions of many leaders in modern psychogenetics without finding a clue or suggestion as to the meaning of these reactions, much less a complete statement of them; as though the problem were nonexistent, or, at least,

unimportant. But it is existent and it is important, for the number of these patients is legion, and they are very sick persons who are entitled to the best care that one can give them. Interpretative psychiatry, therefore, owes a lasting debt to psychoanalysis, which has enabled it to appreciate the tremendous rôle played by fantasies, especially as a source of emotional imbalance, and to understand these paraphilias as symbolic expressions of a conflict, for otherwise these reactions appear to be wholly incomprehensible. One may understand, perhaps, how a person would derive satisfaction from inflicting pain on another, although it is not clear how this act could become so tremendously obsessive and a source of sexual gratification. But what ordinary human logic could explain satisfaction derived by a person from inflicting pain on himself? For this violates the established biologic canons that animals seek to avoid pain, reflexes having been erected to protect the animal from pain. And what is more significant is that it is not general pain that is sought, but a highly specialized form of pain under very special circumstances.

After elucidating the general nature of the problem, orthodox psychoanalysis began to concern itself primarily with further theoretical formulations, but it remained for Stekel to give a clearer basis of reality to the problem by bringing forth a vast amount of case material from which proper concepts could be correctly deduced. This is as it should be: not theories in search of facts, but an abundance of facts from which concepts logically follow. One cannot help but admire the clinical skill with which Stekel elicits significant psychic material that ordinarily lies buried deeply beneath the level of conscious thought, and the brilliant interpretations of dreams, remarkable alike for skill and simplicity. Stekel's material can hardly be said to abound in theoretical contributions; indeed, theories are conspicuously lacking. Such concepts as are presented are based entirely on varied case material, which enhances their value. But although Stekel has gone a long way to clear the problem, he himself admits that the mechanism of the paraphilias, especially that of fetishism, is not as yet fully understood. Much of it still remains to be worked out; the framework only is erected and the safest plan to adopt is to look on Stekel's formulations as hints and suggestions for further and more intensive research.

On the basis of his clinical research, Stekel came to the conclusion that sadism is primary and masochism is secondary. There must be some truth in that, for it was finally acknowledged by Freud. Stekel, however, is so certain of his conclusions that in some cases he merely states that the patient's masochism has developed from an original sadism by way of religion, without troubling himself to prove how it has come about in a particular case. This is to be regretted. Psychoanalysis is still so young a science that it should take nothing for granted or accept anything as established unless supported by fully adequate clinical

research; it is this attitude of drawing wide conclusions from all too slender premises that has brought antagonism from many quarters that otherwise would be sympathetic toward a psychoanalytic interpretation.

Stekel supports his claim that there is a fundamental generic relationship between the paraphilias and the obsessional neuroses by many strong and specious arguments. Many objections can be raised against this theory. A lack of proper balance between love and hate trends is, of course, common to both; but this is true of all neuroses and, for that matter, of many functional psychoses (compare, for instance, the outward masochistic behavior of the depressed manic with his grotesque sadistic fantasies). The death clause, which Stekel submits as characteristic of obsessional neuroses, is also found, he believes, in the paraphilias in question, disguised, according to him, as hatred. This, however, seems to be stretching the point too much, for if hatred parallels the death clause because it is deadly, then, by similar logic, anxiety neuroses should also be included in the argument, for the feeling of guilt in the latter group arises from hatred and death wishes followed by a feeling of guilt and fear of retribution. There is no doubt that there is something that binds all these groups into a close genetic relationship, but what it is is not yet known, and one must wait for further research.

There is, of course, much more to the argument that there is a relation between these paraphilias and homosexuality, for on the one hand homosexuality, too, is an attempt to escape the opposite sex at all costs, and on the other hand homosexual trends are definitely observed in these paraphilias, just as these paraphiliac trends are observed in cases of homosexuality. This similarity, however, while convincing, is not sufficient proof of relationship. Stekel, of course, argues on the premise that biologically and psychically all persons are bisexual and hence that one is overtly or implicity homosexual to the degree that one deviates from heterosexuality. Such an argument is open to many objections, for one may accept the premise of bisexuality and still submit that a person may become turned from the heterosexual path without *eo ipse* leaning over to the homosexual road. A person may become weakened in his heterosexual potency because of a difficult family or social situation, yet not show the least evidence of any homosexual trends, remaining essentially heterosexual, as can be proved by the fact that when the particular inhibitions are removed full potency returns. To whatever extent one may be convinced of the truth of this tenet, its adoption as an unquestioned principle carries with it the danger of a religious dogma and is death to further dispassionate scientific inquiries.

Throughout his work Stekel writes of the significance of constitutional factors in the development of neuroses (his almost invariable phrase is "constitutional factors being granted"), but nowhere does one find a more complete elucidation of the problem. Here and there one

reads of suggestive physical findings, but these are meager and wholly inadequate for the establishment of a premise. Perhaps a complete explanation of this problem is too much to expect of one person, and Stekel has probably done his full share by his exploration of the domain of the instincts and emotions proper. Freud, too, against much opposition in his own ranks has long maintained that constitutional and somatic factors are present in neuroses. The same is true of psychically conditioned physical phenomena. Thus, in one case cited, Stekel writes of a patient who at coitus always experienced a severe cramp simultaneously with orgasm. What is the meaning of this cramp? In another case, he writes of the presence of respiratory difficulties (sensations of choking, asthmatic symptoms, dyspnea, etc.) as expressions of strangling and choking fantasies. This is interesting and suggestive, but it would be desirable to have more data on psychic states as causative factors in physical phenomena. In all, further studies on the constitutional make-up in neuroses and the psychic basis of physical phenomena are sadly needed.

With reference to the problem of imbalance between love and hate trends, one may ask: Can it be considered as fully established that love and hate are bipolar opposites? Is one not likely to make a fetish of bipolarity and bisexuality? Is it true that hatred is inverted love? If so, by what particular process is this inversion effected? Further, is it true that sudden hate reactions are due to explosions of repressed sadism? How is sadism repressed? Why should masochism come in the wake of repressed sadism? Is it true that sadism is cruelty toward those one loves? If so, does it mean that those whom one hates, one really loves? If not, what is the meaning of the hatred toward those whom one does not love?

Is it possible that from an unrequited childhood affection there should spring a hatred so strong as to furnish an emotional drive for many years? If one comes to hate one's mother because of refused love it is not difficult to understand how such hatred would involve all women, but how under such circumstances this generalized hatred should lead to the opposite, the regarding of women as "substitute divinity," is not clear. How does this combination of Blessed Virgin and prostitute come about?

With reference to the sexual etiology of the neuroses, one may also ask: Is it true that the forbidding of onanism plays such a morbid etiologic rôle? Is incest demonstrable in all cases and is it as important and significant as is claimed? Is it possible that a child can be so impressed by the observation that his mother was frivolous or dissolute that this should later change his entire reaction toward the opposite sex? How does the sexual drive become so grossly distorted? Is it not more likely that there is here an "illusory recollection," the patient for reasons

unknown to the psychoanalyst being unable to effect a proper adjustment but bringing the recollections as a rationalization?

Closely related to incest is the problem of homosexuality, much of which is not clear. One hears statements such as "He wanted to be a woman," or "beating the homosexual within himself." It is not clear how through the hatred of women a man comes to hate the feminine component within himself, this leading to homosexuality. How is one to conceive that in beating himself the patient is punishing or attempting to drive out his homosexual component? While other explanations are perhaps equally unsatisfactory, the explanation given is not entirely credible; at least it is not adequately supported. How does a man become a masochist by "symbolically subjecting himself to the woman within himself?" How does the patient's homosexuality form "the basis for his scorn of women, which he transforms into an apotheosis of women"?

If it is at all possible, for whatever reason or whatever causes, to renounce sexuality, does not this suggest that sex is not so strong a drive as is claimed? If social taboos can inhibit sexual activity, then is not the social instinct stronger than the sexual instinct? The problem of sex is important in neuroses, perhaps not because sex is the all-dominant force in life that it is claimed to be, but because in a small number of persons the problem of sex is not adequately balanced, and these are the persons in whom the paraphiliac neuroses develop. They show a precocious sexuality that manifests itself with remarkable strength.

But even though the problem of sex is overemphasized, one must be grateful to psychoanalysis for having called attention to the fact that the problem exists. In the works of other psychiatrists (such as Kraepelin and Janet) the problem of sex is given hardly any consideration.

Throughout Stekel's works, and this is true not only of his writings but of those of many other psychoanalysts, mechanisms are discussed, but the particular processes by which these are effected are given but slight mention. The differentiation between hysterical and epileptic convulsions is not clear. Does Stekel mean that fundamentally these have the same mechanisms? Reversal of relationships is often spoken of, as when the man who as a boy was sickly and was carried a great deal in his mother's arms now fantasies carrying a woman (the mother) in his arms. How does such reversal come about? Dissociation is often mentioned ("The hatred is split off and is carried to the entire sex"). Assuming this to be true, what is the process by which dissociation is effected? Regression to childhood ("I will be a child again") is often mentioned. How is this regression accomplished, and what are the forces that support it? How does the patient symbolize "coddling the child in himself?" The feeling of guilt is often emphasized. Is this feeling as strong as is claimed? While admittedly it appears to play a significant rôle, little is said of its development. The escape or transition

from pain into pleasure is not clear. The death instinct is stated to be the bipolar opposite of the life instinct; but this appears to be a sheer assumption in support of which no data or proofs are submitted.

In spite of all these gaps to be filled, the striking rôle played by psychogenic factors emerges as of tremendous significance. It is obvious, however, that one is only at the threshold and needs more data.

In the many cases presented by Stekel a discussion of social factors is almost lacking; at least it is not evident that these play any particular rôle. This is to be regretted, for social factors seem to play a determined rôle in the etiology of many neuroses, and a study of these factors should be of fundamental importance. But, as though to support the idea that the constitutional factor is large in the etiology of neuroses, one finds that these patients come from both the lower and the upper social strata, from both the city and the country, from homes in which the family situation is bad and also from homes that are almost ideal in every respect, just as in infantile paralysis or other infectious diseases. Psychogenesis? Yes, but working on a soil predisposed to disease.

On some points Stekel appears to be taking issue with Freud, but one gets the impression that, in some instances at least, he merely tries to create differences. Although he criticizes the use of the term libido, he nevertheless uses it frequently himself, writing of promoting the conquests of the libido, etc. According to Freud, these patients use what he calls "screen memories," in which an important occurrence is in the shadow of a harmless one; but Stekel believes that the patient creates important occurrences out of unimportant events. This, it seems, is a distinction without a difference.

A few inaccuracies or, perhaps, unclear statements may be pointed out. With reference to a certain case, Stekel states that the patient's paraphilias could not be located in his dreams and described him as being a hypocrite in his unconscious. Is it possible to fool the unconscious? Again, he describes a case of orthopedic fetishism in a patient whom he psychoanalyzed within ten weeks. Assuming that he had daily sessions, this would make only seventy sessions. It seems hardly credible that even Stekel could adjust so difficult a case in so short a time, especially the heterosexual adjustment which he effected.

Although Stekel writes only of sadomasochism and fetishism in neuroses, the application of the knowledge gained to the problem of psychoses is significant. The irritability of the manic patient and the hate reactions of the paranoid schizophrenic patient are but few of the many expressions of lack of balance in the hate-love reactions in psychoses. In spite of the ingenious arguments advanced by Freud in regard to homosexuality as the basis of paranoia, homosexuality alone does not explain the terrific hate reaction observed in paranoiacs and paranoid persons. Paraphiliac phenomena are found in great abundance in psy-

choses if one wishes to look beneath the surface. The Christ complex in fetishism finds its parallel in the schizophrenic's transformation into Christ. The incest root found in every case of sadomasochism and fetishism is also conspicuous in psychoses, sometimes even on the surface. Sex life in psychoses, especially in schizophrenia, is less exuberant and more disorganized than in neuroses. In schizophrenia, as in fetishism, the patient discards the woman, but instead of substituting the female by a symbol he escapes into paraphiliac fantasies. Both are engaged with themselves while apparently seeking the object of their love outside themselves. Here is a fertile virgin soil to which clinical research could be directed with great profit.

Many physicians, not excepting practicing psychiatrists, adopt a wrong attitude toward the paraphilias, and when the question of treatment comes up usually dismiss the problem by the statement: "This man is a pervert; there is nothing that we can do about it." However, by stigmatizing the condition as a perversion one does not solve the problem, but merely gives vent to a sense of moral indignation that has no place in professional work. These patients should be regarded neither as perverts nor as degenerates, but as sick persons whose sickness is derived from environmental sources on the basis of arrested development (or perhaps maldevelopment) in the biologic and psychic evolution of man. They are no more responsible for that than a patient is responsible for having a particular disease. Rather than speak of these paraphilias as perversions or degeneracies it is much more proper to regard them as neuroses, for, as in neuroses, one is often able to trace much of the reaction to emotional factors which can be analyzed for their individual components. While they appear to differ from neuroses in that they are established psychosexual reactions, one must remember that even in neuroses paraphiliac trends are often observed, and the line of demarcation is not so sharp and clearcut as it might appear. On the other hand, one has no difficulty in observing in these paraphilias a large number of hysterical and obsessional features, so that by any definition these paraphilias are neuroses and should be treated as such. In disagreement with Freud, who believes that paraphilias are not reducible, Stekel maintains that some of these are curable, as his case material well shows, and that in many cases improvement, at least, can be effected. This is a decided advance. The fact that there still remain a large number of cases in which the paraphilia cannot be cured does not by any means signify that it is not curable, but merely that the proper avenue of approach has not yet been found. In the future, psychotherapy will have to devise other methods and other technics for treatment in these cases.

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Abstracts from Current Literature

COLLOIDOCHEMICAL ASPECTS OF SENILE AND PRESENILE TISSUE ALTERATIONS: THE HYSTERETIC SYNDROME AS A TYPE OF CEREBRAL REACTION. A. VON BRAUNMÜHL, Ztschr. f. d. ges. Neurol. u. Psychiat. **142**:1 (Sept.) 1932.

Braunmühl's fundamental contribution is written in honor of the sixtieth birthday of F. Ast. The author points out that morphologic contributions on the senile changes in the brain have left little to be desired, whereas the explanation of such structures as senile plaques, corpora amylacea and fibrillary changes is yet in its infancy. He attacks the problem from the standpoint of elementary and well defined processes known to colloid chemistry. Taking up first the senile plaque, he states that this peculiar structure, with its nucleus, open space and corona, is one of the most notable features of precipitation in colloid chemistry. There is sometimes a little confusion because of the absence of clear space or double nuclei, but by means of diagrams and even models in which he has produced plaquelike pictures in woven cloth, he has brought out the essential nature of these structures. Moreover, in certain cases, the finer details of radial and tangential formations within the plaques give a much better clue to their origin. In some instances, even, these plaques assume an appearance suggestive of Liesegang's rings; the substances therefore represent precipitates in the tissue, and the neural and glial structures have nothing to do primarily with the genesis of the plaque. It seems that there is a precipitation of a colloidal material from a supersaturated solution by an electrolyte. For this to occur there must be circulating within the brain a certain amount of waste material that can be deposited. But the number of the plaques apparently does not run parallel to the amount of fundamental substances, since two factors determine the precipitation: the presence of the material and certain optimal conditions for this precipitation.

Corpora amylacea are in many respects similar to senile plaques in their intimate structure and can be understood only on the basis of colloid chemistry. The successive concentric layer formation betokening rhythmic precipitation is exactly what one finds in Liesegang's rings. The same holds for the concretions of similar concentric type so often found in the brain.

The cellular alterations characterized by the presence of granules and vacuoles also probably correspond to certain well known features of colloidal precipitation; indeed, one sees the dark granule surrounded by a lighter halo, which indicates colloidal separation phenomena with a reduction to an aggregated phase in the center and a dispersed phase at the periphery, which may well be spoken of as syneresis.

The fibrillary condensation first described by Alzheimer has received many explanations based more on vital than on chemical phenomena. In studying the living nerve cell, the cytoplasm is found to be practically uniform, even on dark-field examination, and no neurofibrils are apparent. However, if the cytoplasm is penetrated by a needle, a fine fibrillary network becomes evident, and any other damage to the nerve cell brings to light the characteristic endofibrillary structure that is so well known in cerebral cytology. According to Péterfi, there is a manifestation of salting out, or precipitation of a living colloidal substance of nerve fibers which is associated with dehydration of the micellae. The colloids of the nerve cell, therefore, evidently have a tendency for the molecules to become oriented in a linear fashion; this, combined with the tendency to eliminate water in the process of coagulation, brings about the foundation of the histologically visible fibrils. "The neurofibril structure of living nerve tissue can therefore be thought of as a latent structure." Observations on the invertebrates by Stöhr,

Cajal and others and experiments by de Renyi confirm the fundamental fibrillary foundation in the nerve cells but indicate that it is a latent rather than an evident structuralization. When the living nerve cells are subjected to adverse biochemical situations they often respond in peculiar manners, with swelling of the axis-cylinders and tortuosity and agglomeration of the elementary fibrils. The fibrillary changes within the ganglion cells are therefore probably to be explained on the colloidochemical principle of imbibition, with certain changes in optical properties and also in the propensities for taking up the silver molecule from alkaline solutions. The author has constructed a model of rubber bands dipped in benzine to demonstrate his point.

Argentophilic granules (*Kugeln*) furnish the touchstone in explaining the colloidochemical aspects of the problem in the senile brain. The explanation for these peculiar structures has been varied, but always based on a morphologic or biologic foundation. There can be no doubt, however, that these balls bear a close genetic relationship to the micellar apparatus of the nerve cell and to its fibrillary nature. Their completed structure indicates their origin from the rolled up and contracted threadlike structures of the nerve cell. Similar pictures can be obtained by centrifugation and by tearing out of the nerve cell its delicate fibrillary structure. Whether argentophilic balls or fibrillary condensation shall occur depends on the tempo and the intensity of dissolution. In addition to hysteresis, the opposite change may take place in the micellar units, which undergo a complete separation with the formation of a gel-like substance, partially or completely coagulated; secondarily, the precipitated mass may become hydrated again sufficiently to approach the colloidal dispersion phase and finally to go over into the watery condition which is observed in Pick's disease as rounded water vacuoles of hydropic degeneration. There must be in this manifestation a reversible reaction, sol to gel and gel to sol, with changes, naturally, in the affinity for the silver ion. The ballooning of the cell takes place only when the micellar portions imbibe water and undergo hydration. Both hysteresis and syneresis may therefore be present at the same time but be taking place in different degrees.

Turning to a discussion of the peculiar glia cells of Pick's disease, the author finds here also indications of colloidochemical activities, particularly since there is a marked quantity of iron in these cells. The cells contain both fat and iron, but if the sections are washed with strong acid before staining, the fat no longer shows the characteristic granular picture, indicating that the iron is in some way bound to the fat. The author believes that the colloidochemical disturbances in the glia cells homologous to those in the nerve cells are responsible for these peculiar pictures. The iron probably acts as a vital dye and on reaching a certain concentration within the cell vacuole undergoes precipitation. The occurrence of iron in granules, rather than diffusely, is another argument in favor of the vital character of the process, since the dead cell is recognized by its diffuse staining with the vital dye.

The damage to the myelin sheaths in Pick's disease and in other conditions has been described from the standpoint of morphology, but there is no good understanding of its nature from the colloidochemical standpoint. There is no doubt that the foundation for it occurs during life, although the manifestations may develop only after the use of coagulating substances. The swelling, vacuolation, irregularity and condensation give every indication of a disturbed water balance in the myelin sheath, and the appearance of the finished product undoubtedly depends both on the phase and on the rapidity with which the phase is changing. "In the realm of physical chemistry of colloidal solutions, of which this phenomenon is certainly a manifestation, the relationships between solvent and solute are particularly intimate." The effects of ionic concentrations, temperature and acidity influence the eventual result. Other phenomena of similar character occur probably in the intercellular and interfibrillar tissues, giving the morphologic appearance of status spongiosus and status cribratus; both of these are frequently associated with the other colloidochemical changes previously mentioned. Histopathologists

again have used vitalistic explanations, but underlying the whole condition is probably a disturbance in the mechanism of the lymph circulation and alteration of the distribution of water between the tissues and the vascular walls. The author believes that at least one variety of status spongiosus results from hysteresis with dehydration and that such a process may occur relatively suddenly and occasionally in stormy fashion, although he is not ready to admit that its development is correlated with the clinical picture of fits and spells.

In status cribratus the fundamental disturbance is apparently slowing of the circulation, which in turn unbalances the diffusion of fluids between vessels and tissues. Here again from the physical standpoint there is probably a change of the water from the bound to the free state, with actual dehydration of the tissue elements.

Discussing the factual material heretofore presented, the author speaks of ageing in one or another of the body systems as a change in the total organism from the structural or functional aspect and says that the problem of involution is a constitutional one. With ageing of the body colloids, there is a certain dehydration, with a tendency from dispersion to aggregation recognized under various terms as an entropic phenomenon, protoplasmic hysteresis leading to physiologic death. The diminution in dispersion (condensation), with dehydration, brings about the salting out from solutions of slaglike material which is deposited in the tissue along with an excess of free water. It is thus that the hysteretic principle is manifested by morphologic methods. Hysteretic processes cannot be primarily analyzed, but are manifested by certain detectable secondary alterations in which phenomena of solution and precipitation are the most prominent. The author meets the problem of senile dementia without plaques or fibrillary condensation by postulating the presence of the primary alterations without conditions leading to the secondary or precipitating manifestations that are found in the ordinary case. He invokes the primary hysteretic alterations in the tissue which are as yet invisible and which, if continued long enough, will result in the visible secondary manifestations. "The hysteretic principle or syndrome is a general form of reaction indwelling in the brain which under physiologic and especially pathologic conditions can become manifested." Under physiologic conditions, for instance in hibernating reptiles, fibrillary changes can be found in the ganglion cells, which obviously speak for a reversible (hydration-dehydration) alteration.

Coming to the heredodegenerative diseases, the author finds morphologic and biochemical manifestations of his hysteretic principle, although not conforming as exactly as the involutionary process; certainly the ballooning of the cells in Tay-Sachs' disease is a colloidochemical disturbance, while the monster cells in Wilson's disease betoken similar alterations. The cells of Huntington's disease resemble those previously described, while the alterations in amyotrophic lateral sclerosis, Friedreich's disease and cerebellar degeneration seem to conform less closely with the phenomena previously described. The author is in complete disagreement with Schaffer in considering that imbibition by the hyaloplasm is a primary and fundamental phenomenon.

In regard to the heredodegenerative diseases characterized by local atrophies and abiotrophy, a partial hysteretic syndrome is probably the most adequate explanation.

In recapitulation, the author mentions a number of tissue changes in the ageing brain which are apparently secondary facultative manifestations of a primary disturbance in a colloidochemical balance. The morphologic structures are to be considered as results of solution and precipitation, of hysteresis and syneresis. The hysteretic principle or syndrome is an indwelling general type of reaction, a central nervous system "modus," which occurs under various physiologic and pathologic circumstances. Finally, the author brings all the abiotrophic diseases into one group with the senile alterations as different manifestations of the same hysteretic principle.

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COMPARATIVE ANATOMY OF THE NUCLEI SURROUNDING THE AQUEDUCT OF SYLVIUS. DONAL SHEEHAN, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **35**:1, 1933.

Sheehan discusses the masses of cells between the decussation of the trochlear nerves and the posterior commissure. In the orang-utan, four cellular masses can be identified in this area: (1) the retrotrochlear region, (2) the region around the trochlear nucleus, (3) the region around the oculomotor nucleus and (4) the region between the most ventral portion of the oculomotor nucleus and the posterior commissure. Disregarding the outposts of the nucleus paramedianus dorsalis and of the nucleus angustus (Ziehen), there is on the mesial surface of the retrotrochlear region a sharply outlined nucleus consisting at first of small cells and later of larger cells, which become more and more numerous cephalad. This nucleus is found in the supratrochlear region. Similar cells exist mesially, producing together with the supratrochlear cells a "fountain-like" nucleus. These cells become diminished in number at the ventral end of the nucleus trochlearis and terminate in the region of the oculomotor nucleus. This fountain-like nucleus contains numerous smaller cells which are indistinguishable from those designated by Ziehen as the nucleus parvicellulatus. These small cells can be seen throughout this entire region, except where the fountain-like nucleus is unusually well developed. In the retrotrochlear region the small cells assume the form of a nucleus which runs alongside the nucleus tegmenti dorsalis of Gudden. The latter cannot be distinguished in all animals from the retrotrochlear cells in the region dorsal to the posterior longitudinal fasciculus in the pons.

The fountain-like nucleus can be identified in all animals, but its development varies in the different species. The variations occur in (1) the number of cells and (2) in the appearance of the dorsal group of small cells situated more ventrally around the aqueduct of Sylvius (in Ungulata, Edentata and Marsupialia), and (3) in some animals (Ungulata), as the nucleus extends cephalad it appears as one cellular mass, whereas in other species it appears in the form of branches arising from a median column which become richer and richer in cells as they spread laterally, forming eventually a separate collection of cells; this is especially the case in man, in whom the relation between the mesial and the lateral portions is not so marked as in apes. A well developed fountain-like nucleus is also present in Carnivora. In Natantia, the nucleus is characterized by a greater number of small cells in its mesial portion. It is also noteworthy that in Marsupialia the nucleus bears a certain resemblance to that in higher mammals.

The second nucleus in this region is designated by Sheehan as the nucleus dorsolateralis, because it is located dorsolateral to the fountain-like nucleus and the fasciculus longitudinalis posterior. This is not the same structure which is designated by other observers as the nucleus lateralis aqueductus or anuli aqueductus. The nucleus dorsolateralis consists of sharply outlined groups of cells situated first laterad to the aqueduct and more orally in the retrotrochlear and trochlear regions. Here too the cells are large, with small cells interspersed between them, especially in the area between the aqueduct and the fasciculus longitudinalis posterior, where they form a sharply outlined accumulation of cells constituting in some animals, with a poorly developed nucleus dorsolateralis, a distinct and separate group of cells. These cells, however, can be readily differentiated from the nucleus parvicellulatus.

The nucleus dorsolateralis shows great developmental variations in the different species. It is best developed in the orang-utan and other apes; in Carnivora, it consists of two sharply isolated groups of large cells; in Ungulata, the nucleus is strikingly parvicellular. The nucleus may be regarded as an expression of higher cerebral development, because the lower the animal in the scale the poorer the nucleus is developed. Its greatest development is found in the oral portion of the trochlear region. It can be traced, although not entirely, into the oculomotor nucleus. It is important to distinguish the nucleus dorsolateralis from the locus caeruleus and from the cells regarded by Kohnstamm and Quensel as parts of the nucleus Darkshevichii.

The nucleus parvicellulatus, according to Sheehan, is not a nucleus in the true sense of the term. It may be said to be represented by diffuse small cells which are interspersed between all the previously described nuclei. These cells are predominatingly laterad and dorsad to the aqueductus Sylvii and are less dense mesially than laterally.

The nucleus Darkshevichii is better known as the nucleus fasciculi longitudinalis posterioris; it is situated at the dorsolateral margin of the fasciculus longitudinalis posterior at the point where the nucleus oculomotorius has reached its highest development and where it extends into the region of the commissure. The nucleus Darkshevichii shows relatively wide variations in size and is unusually well developed in the lower mammals.

Sheehan suggests that all these nuclei may possibly be vegetative centers, although he admits that thus far no satisfactory proof has been adduced in substantiation of such a hypothesis.

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PAIN AND TENDERNESS OF ABDOMINAL WALL. J. B. CARNETT, J. A. M. A. **102**:345 (Feb. 3) 1934.

According to Carnett, pain and tenderness occur far more frequently in the abdominal wall than in the abdominal viscera. Palpation over relaxed abdominal muscles fails completely to differentiate parietal from visceral tenderness. Parietal tenderness and, inferentially, pain are demonstrated best by firm palpation while the patient balloons out the abdomen and holds the abdominal muscles as tense as possible. Hypersensitiveness either on pricking or stroking the abdominal skin with a pin or on pinching a liberal fold of skin and fat indicates parietal tenderness. Pain and tenderness of the abdominal parietes are variously interpreted by different diagnosticians. The majority fail to consider the possibility of these two symptoms being located in the abdominal wall, neglect to apply the special tests to determine their parietal location and wrongly ascribe them to an existent or nonexistent lesion of an underlying viscus. Many times when the pain and tenderness are not recognized as parietal they are attributed to malingering, hysteria or some other vague neurosis because they are atypical of organic visceral disease or the latter has been excluded by extensive clinical and laboratory studies, often including one or more futile laparotomies. The four areas of distribution of the first lumbar nerve afford many points of interest in the study of parietal pain and tenderness. Libman's classification of patients into sensitive and hyposensitive in accordance with his studies at the mastoid tip and styloid process is not applicable to cases of pain and tenderness of the abdominal parietes. The only parietal pain and tenderness of fairly consistent type in location, size and frequency of association with a visceral lesion of which the author is aware are those observed in cases of gastric or duodenal ulcer in an area the size of a thumb nail situated in the midline about midway between the umbilicus and the tip of the xiphoid process. It is his belief that in biliary colic pain and tenderness referred to the shoulder or scapula occur only as an aggravation of neuralgia primarily due to some extraneous cause and that the neuralgia is not relieved by cure of the pathologic condition in the biliary tract. Parietal pain and tenderness of the abdomen are usually due to neuralgia and, as a rule, are independent of any intra-abdominal lesion. Failure to recognize neuralgia by the pinch test of skin and fat leads to many erroneous diagnoses. Pain and tenderness may not always be due to the same mechanism, a fact which seems to have escaped many clinicians. Chronic parietal pain and tenderness that have been present for months or years are not due to and are not cured by the operative removal of intra-abdominal lesions. Chronic parietal tenderness in the absence of spontaneous pain is a not infrequent observation in routine physical examinations on patients with spinal lesions causing mild neuralgia. Chronic neuralgia may start in the chronic form, but more commonly it is preceded by recurrent acute attacks which are frequently mistaken for acute visceral disease. Acute parietal pain and tenderness are usually due to acute infections that do not require operation and disappear rapidly on

subsidence of the toxemia resulting from the infections. Acute pain and tenderness of the abdominal wall, and not infrequently elsewhere on the body surface, are caused by acute appendicitis infrequently, by acute pelvic inflammatory disease fairly commonly and by other intra-abdominal infections rather rarely. Acute toxemia is responsible for the great majority of the cases in which acute intra-abdominal infections cause acute parietal pain and tenderness, especially when the affected parietal area lies entirely outside the Head zone for the particular visceral lesion present. Most commonly, acute pain and tenderness of the abdominal wall are not associated with an acute intra-abdominal lesion, and, on the other hand, acute visceral lesions commonly do not cause parietal pain and tenderness. Acute neuralgia calls for treatment of the causative acute infection or spinal injury. Scoliosis is treated by appropriate exercises and by elevation of the heel of the shoe if a short leg is found. Parietal pain and tenderness due to spinal arthritis are promptly relieved in the majority of cases by mild irradiation of that portion of the spine from which the affected nerves emerge. Treatment of excessive lumbar lordosis by the Goldthwait special exercises to correct bad body mechanics cures parietal pain and benefits digestive disturbances, although the tenderness may be slow in disappearing.

EDITOR'S ABSTRACT.

INNERVATION OF THE LARYNX: III. EXPERIMENTAL PARALYSIS OF THE LARYNGEAL NERVE. FREDERICK LEMERE, Arch. Otolaryng. **18**:413 (Oct.) 1933.

All laryngeal muscles are innervated unilaterally by the recurrent laryngeal nerve except the cricothyroid muscle, which is supplied by the corresponding external ramus of the superior laryngeal nerve. As the interarytenoid is a midline muscle, it is innervated by both the right and the left recurrent laryngeal nerve. Faulty technic led Exner to think that each laryngeal muscle was bilaterally and doubly supplied by both the superior and the recurrent laryngeal nerves. Luschka believed that the interarytenoid muscle is innervated by both the recurrent and the superior laryngeal nerves. This idea is widely held at present. The author believes that the branches of the internal ramus of the superior laryngeal nerve, which appeared to innervate the interarytenoid muscle, in reality pass on through to supply the underlying mucous membrane. He records changes in the glottic picture following experimental paralysis of the laryngeal nerves. The cricothyroid muscle stretches the corresponding vocal cord and also tends to adduct it. The thyro-arytenoid relaxes the vocal cord and also adducts it. The lateral crico-arytenoid adducts the anterior or membranous half of the cord. The posterior crico-arytenoid adducts or swings the vocal cord out laterally, dilating the glottis on the same side. The interarytenoid closes the cartilaginous or posterior half of the glottis. The functions of the larynx are protection in swallowing, the maintenance of a patent airway and vocalization. In quiet breathing, complete denervation or death the cords assume the cadaveric position.

Sectioning of both recurrent laryngeal nerves causes paralysis of all the laryngeal muscles except the cricothyroid, giving partial adduction, midway between full adduction and the cadaveric position, which has always previously been associated with this type of paralysis. The partial adduction, together with the absence of normal inspiratory abduction resulting from paralysis of the posterior crico-arytenoid muscles, causes the most marked dyspnea found in association with paralysis of the laryngeal nerves. It may immediately result in death, especially if the glottis is small. Vocalization is impossible or coarse, but coughing occurs.

Sectioning of one recurrent laryngeal nerve causes some dyspnea and only a little hoarseness; swallowing is not interfered with; the opposite cord crosses over the midline.

Bilateral sectioning of the external rami of the superior laryngeal nerves has no effect on the glottic picture. The tension of the vocal cords is lost, and they wrinkle on attempted phonation. Unilateral section causes slight hoarseness. Bilateral sectioning of the external rami and of the recurrent nerves results in

complete paralysis of all the intrinsic muscles, with the glottis in the cadaveric position and vocalization impossible. The slightest exertion causes dyspnea. Sectioning of the external ramus of the superior laryngeal nerve on one side and the recurrent laryngeal nerve on the other side causes paralysis of the cricothyroid muscle on one side and partial adduction of the vocal cord on the other side with paralysis of abduction.

Sectioning of both recurrent laryngeal nerves and of the external ramus on one side causes complete paralysis of the cord on the latter side, the other cord being in a partially abducted position.

Sectioning of the external rami of both superior laryngeal nerves and the recurrent nerve on one side results in complete paralysis of the latter side and relaxation of the cord on the other.

This article does not lend itself to being abstracted as it is written in concise form. Those interested should read it in the original.

HUNTER, Philadelphia.

ANATOMIC BASIS OF CLINICAL MANIFESTATIONS OF CEREBRAL VASCULAR DISORDERS. I. J. SANDS, J. A. M. A. **99**:1599 (Nov. 5) 1932.

Sands calls attention to the fact that recent investigations of the cerebral circulation and the discovery of the nerve control of the pial and the cerebral vessels have aroused new interest in the manifestations of cerebral vascular disorders. Pfeifer and Cobb have disproved the old theory of end-arteries in the brain; in fact, they have demonstrated conclusively that the capillary bed of the whole cerebral cortex is an endless network, and that there is a free anastomosis between the capillaries of both the surface and the depth of the brain. Hassin has again called attention to the presence of nerve endings in the pial vessels, and Penfield has shown that the intracerebral arteries are innervated in a manner similar to that of the blood vessels of the pia, and, moreover, that the two nerve plexuses are continuous. Forbes and Wolff have demonstrated that the nerve supply of the vessels of the brain is derived from the sympathetic system, and that stimulation of the vagus causes dilatation of the vessels and stimulation of the sympathetic causes their constriction. These facts are of prime importance in the evaluation of the signs and symptoms of the various cerebral vascular disorders. Cerebral arteriosclerosis is the most common of the cerebral vascular disorders. It has been commonly accepted that the condition occurs most frequently in persons of superior intelligence. If one is to assume that there is a close relationship between excessive function and wear and tear of an organ, it is logical to infer that those who are particularly engaged in continuous cerebration must necessarily draw a large amount of blood to the brain, causing the cerebral vessels to function excessively. Emphasis has been placed on the strain of modern civilization as a factor in producing cerebral arteriosclerosis. The stress and strain of modern life, as reflected in anxiety, worry and emotional tension, must be expressed through the vegetative nervous system, and, consequently, on the cerebral vascular system through the action by the vagus and sympathetic systems. Heredity is regarded as playing an etiologic rôle. Another consideration in cerebral arteriosclerosis is the Virchow-Robin lymphatic system of the brain. While there is still considerable dispute as to the presence of the Virchow-Robin lymph spaces in the adventitial sheaths of the cerebral arteries, many who have worked with neuropathologic material are convinced that such a system exists. If, therefore, cerebral arteriosclerosis is found more frequently in those who pursue intellectual work and especially if it is apt to accompany the stress and strain of modern life, it is reasonable to assume that the lymphatic system of the brain must carry away excessive amounts of catabolic substances that may prove a source of irritation to the vessel wall and may start a train of physical and chemical changes that may lead to anatomically proved arteriosclerosis. People with cerebral arteriosclerosis generally complain of headache, dizziness, ringing

in the ears, a knocking sensation in the head, a fulness in the head and an inability to concentrate. They are usually irritable persons. The sclerotic artery is unable to relax adequately before the oncoming blood stream and transmits the impact of the blood column directly to the adjacent brain tissue. The continuous pounding of the blood stream on the brain tissue through the sclerosed artery causes irritation of the nerve tissue. Moreover, the Virchow-Robin lymph channel, being handicapped by the arteriosclerotic changes in the vessel walls, is no longer as efficient a drainage system as in normal persons, and this may result in retaining toxic and irritating catabolic substances in the brain. The sclerosed internal carotid artery, passing through the petrous portion of the temporal bone, may prove a source of irritation to the cochlear and vestibular nerves, and may be in part responsible for the dizziness, the knocking sensation and the ringing in the ears of which these persons complain.

EDITOR'S ABSTRACT.

A NEW CONCEPTION OF MORAL BEHAVIOR. E. MIRA, J. Crim. Law & Criminol. **24**:860 (Jan.-Feb.) 1934.

In this article an attempt is made to explain moral behavior in terms of psychologic motives. The theories of morality are outlined, and it is pointed out that there are many of them. One postulates an inborn origin for the moral sense or tendencies, and another affirms that their acquisition takes place in the process of individual development. Psychoanalysts favor the theory of unity and derive the origin of all morality from evolution and sublimation of the destructive principles which are directed against (1) the environment, and (2) the self. There are other dualistic theories of the origin of morality, the chief being that of Bergson, in which he asserted (1) that this origin is based in the elements of society, and (2) that it receives its strength from an inner vital impulse. Piaget showed experimentally that there are two types of moral conduct: heteronomous, due to force or external compulsion, and autonomous, based on the existence of mutual respect between the individual person and the community. The problem presented in this paper is how to study morality, assuming it to be an attitude of the person to be studied, in the same way that intelligence or other similar problems are studied. The technic used was to give the subject a printed booklet with instructions regarding the problem and ten possible solutions for it, which are to be classified. The first problem is how a bitter husband should deal with his particular problem. Of a group of randomly selected solutions, 187 of 289 husbands preferred the solution of settling their difficulty by resorting to legal redress, while 185 of 288 women selected private separation. Another problem, one given to trained nurses, was intended to show what decision might be reached as to how medicine should be distributed among a number of patients for whom an insufficient amount was available; this problem was intended to show which patients the nurse might try to save, and the reason for such selections. All responses were compared with those received by other experimenters, and Mira concludes: "1. Moral conduct does not depend upon the existence of any definite general factor or motive. 2. Its causes are more likely to be found in the evolution of emotional attitudes rather than in a process of reasoning. 3. There are no moral standards of judgment that allow of a definite and constant ethical evaluation of all the possible reactions in the presence of definite circumstances. 4. The standard of moral judgment not only varies with persons, but differs in the same person, according to the different kinds of situations faced and the moment in which he makes the judgment. 5. There are certain classes of people that are considered as morally deficient who, nevertheless, in behavior tests have shown themselves more unselfish than those regarded as normal."

Mira discusses all of these questions and concludes that it is the duty of the modern psychologist, however difficult or vexatious it may be, to continue the study of the mysterious enigma called human personality.

SELLING, Detroit, Mich.

STUDIES IN EXPERIMENTAL PSYCHIATRY: IV. DETERIORATION OF "G" IN PSYCHOTIC PATIENTS. CONSTANCE SIMMINS, J. Ment. Sc. **79**:704, 1933.

Psychologic work in psychiatry has usually been based on the so-called faculty theory of the mind, which classified mental processes into various independent faculties, such as memory, perceptions and attention. When a psychiatrist assumes that by measuring one member of a class of mental occurrences he has obtained a measure of every member of the class the consequences are absurd, harmful and misleading. If a patient fails to repeat a series of six digits there is no justification to report that the patient has a bad memory, or if a child cannot solve some tests in the Porteus-Maze series it is fallacious to state that he lacks in foresight. Methods were devised for measuring the amount of correlation between abilities, that is, for measuring the extent to which ability in one performance tends to correspond with ability in another performance. The score obtained by each person in the test can be divided into two parts. One part depends on an element or factor which remains always the same for all the abilities of the same person. This has been called by Spearman "g," for general ability or general intelligence. The other part depends on a second factor, which differs freely from one ability to another, and this is called "s." In other words, whereas previously an ability was conceived and then measurement was sought, now one first makes the measurement and then tries to figure out what ability it may represent. The three mental processes by which intellect is advanced are: 1. The apprehension of experience. A person tends to be aware of what goes on in his own mind. 2. The education of relations. When a person has in mind any two or more items, he has more or less power to bring to mind any relation that essentially holds between them. 3. The education of correlates. When a person has in mind any item together with a relation, he has more or less power to bring to mind the correlative idea.

"G" is concerned with education of relations. "G" can be measured best by nonverbal tests, which have been devised by various psychologists and were specially combined in this study by the author. Two hundred psychotic patients were tested for present g-ability by means of present perceptual g-tests; g-ability before the onset of illness by means of vocabulary tests, and memory for recent impressions of words, numbers and pictures. There is evidence that both the incidence and the amount of g-deterioration agree with the degree of illness. This varied from 70 per cent in a hospital for chronic cases to 15 per cent in a hospital for acute cases. There was no g-deterioration in patients after recovery, in patients with psychoneurosis, in two patients with "puerperal psychosis" and in a special patient with dementia praecox whose p-score was between 1 and 4. The author believes that the measurement of g-deterioration isolates a factor in the patient's ability to deal effectively with the external world or with the internal world of the patient's experience. It is hoped that the measurement of the g-deterioration may be correlated with the nature of the cortical organization.

KASANIN, Howard, R. I.

DEHYDRATION IN EPILEPSY. J. L. FETTERMAN, J. A. M. A. **100**:1005 (April 1) 1933.

In view of the conflicting opinions on dehydration in epilepsy, Fetterman and Kumin report their clinical experience with the dehydration treatment of twenty-one epileptic patients. Nine patients were hospitalized, each for a period of about a month. The daily free fluid intake was started at 1,000 cc., and reduced by 100 cc. each day or on alternate days, until the patient was allowed to have but 400 to 500 cc. The twelve ambulatory patients were placed on a similar dehydration program. They were advised, after a gradual reduction, to limit their free fluids to 500 cc. daily. In addition, most of them were put on a diet with a predominant acid ash. The nine hospitalized patients presented cases of chronic epilepsy; most of them had been having frequent seizures over a period of years. The course of their illness had been little influenced by earlier therapy. A brief trial of dehydration made little change in their status. It is noteworthy that a change

in the regimen from dehydration to hydration induced seizures in one subject but did not precipitate attacks in the others. Also, the withdrawal of phenobarbital from those on a dehydration program induced attacks regularly. There was no striking change in the general health of the patients during dehydration. All complained of thirst—a few, quite bitterly. The others became readily accustomed to the low intake. The specific gravity of the urine rose. Acetone appeared occasionally. Four of the nine patients continued on a regimen of dehydration following the stay in the hospital; one showed no change in the number or severity of attacks for a period of six months; another likewise was not benefited by the low intake of fluid; one patient was decidedly relieved for three months, only to suffer a return to her usual periodicity of seizures, and the other one was improved during dehydration, but there was further reduction in attacks when unlimited fluids were allowed. The twelve patients included in the ambulatory group were likewise chronic epileptic subjects whose seizures were frequent and had been present for a period of years. Earlier therapy had not benefited them substantially. During the period of dehydration, there was no marked alteration in the general health or in the frequency of the seizures. One patient, a girl, aged 15, whose epilepsy developed after an attack of encephalitis, responded well to the treatment with phenobarbital and dehydration. The attacks stopped for eighteen months. When the dehydration was discontinued, there were no attacks, but when the phenobarbital was discontinued, the attacks returned. The benefit that this patient enjoyed was therefore to be attributed more to the sedative therapy than to the dehydration.

EDITOR'S ABSTRACT.

GLIOMA OF THE SEPTUM PELLUCIDUM. SALVATORE MARRAS, Riv. di pat. nerv. **42**:265 (Oct.) 1933.

The author describes a case of primary glioma of the septum pellucidum. Primary tumors of this region are rare, and there is a lack of detailed histologic study in the few cases reported in the literature. The histologic diagnosis in Marras' case was glioma of a mixed type, varying from unipolar spongioblastoma to fibrillary astrocytoma. The literature indicates that all primary tumors of the septum pellucidum have been gliomas.

The clinical picture presented in the various cases reviewed differed somewhat in each case; in some cases no symptoms had been recorded during the life of the patient, the tumor having been found at autopsy. Occasionally, mental symptoms, such as disorientation, slight confusion, amnesia and bizarre fixed attitudes, have resulted from the growth. In one case the mental picture simulated dementia paralytica. Neurologically, in one case described by Bailey there were apraxia, generalized myoclonia and visual disturbances.

In the case described by the author there were bilateral optic neuritis, a slight convergent strabismus, slight contracture of the upper part of the arm, slight nystagmus, hypokinesis of the face and vasomotor changes, with a papular eruption on the face. Later, a fever developed. A roentgenogram showed considerable enlargement of the sella turcica. Spinal puncture disclosed high pressure of the cerebrospinal fluid. There were insomnia and generalized pain in the legs, with increased deep reflexes. Two periods of unconsciousness were noted, the last being accompanied by convulsive seizures. At autopsy, the tumor was found to originate from the septum pellucidum, to develop underneath the anterior portion of the corpus callosum and to protrude in both lateral ventricles.

The author believes that compression of the tumor against the basal ganglia was the cause of the contracture and paresis, that the loss of equilibrium and nystagmus were probably cerebellar symptoms due to intracranial hypertension, and that the loss of consciousness and convulsive seizures were a more direct result of increased intracranial pressure. He does not correlate any symptoms with the involvement of the septum pellucidum, though this structure appeared quite distended and thin.

FERRARO, New York.

THE STATE OF THE BLADDER AND THE SPHINCTERS IN COMPLETE TRANSVERSE LESIONS OF THE SPINAL CORD AND CAUDA EQUINA. D. DENNY-BROWN and E. GRAEME ROBERTSON, Brain **56**:397 (Dec.) 1933.

In reviewing pertinent literature it was found that the integrity of the pelvic nerves appears of paramount importance in the adequate performance of normal "voluntary" micturition. Complete destruction of the lumbosacral segments of the spinal cord and complete transverse section of the cord above the lumbar segments cause atony of the bladder, later followed by a state of automatic micturition. Three patients with clinical evidence of lesions of the cauda equina and three patients with transverse lesions of the spinal cord form the basis of this important investigation. Vesical pressure was recorded by a special technic which permitted the registration of the discharge through the sphincter.

Distention of the bladder produced contraction of the detrusor urinae elicited by stretch of that muscle, and this is evoked only by increment of stretch. Increase of passive pressure on the bladder via the abdominal wall similarly evoked contraction of the detrusor. A peripheral reaction of reflex character must be postulated to account for this. A certain degree of active reaction to distention seems to be an adequate stimulus for the setting up of discharges of the whole detrusor mechanism by a reflex of the sacral spinal segments. Reflex facilitation of micturition may be enhanced by cutaneous stimuli from the sacral segments and merges with the effects of the "sacral reflex." The internal sphincter reflects a degree of contraction reciprocal to that of the detrusor at the time and is not under separate control. Complete spinal lesions show three fundamental states of disturbance in micturition: 1. A depression of function with an inactive detrusor and a tonic internal sphincter is the transient immediate result of a sudden deprivation of innervation. 2. Incontinence during straining movements characterizes the isolated survival of the peripheral plexus. 3. A more facile and complete development of automatic micturition, coordinated with defecation and subjected to the interplay of spinal reflexes, is the expression of the reflex behavior of the isolated sacral segments. To understand the various detailed transitions in the development of automatic micturition, a careful reading of this excellent article is necessary.

MICHAELS, Boston.

DEMENTIA PARALYTICA IN KOREA. K. KUBO and R. HATTORI, Folia psychiat. et neurol. japon. **1**:10 (Dec.) 1933.

In a previous statistical study (1931) the authors reported a higher incidence of dementia paralytica among Japanese living in Korea than among Koreans. Tabes dorsalis seems to occur seldom in Koreans; only two cases were observed by the authors during the last eight years. There was no increase in the hospital admission rate for Koreans with dementia paralytica in recent years. Only a slight difference was noted in the number of ambulatory cases of systemic syphilis in Koreans and in Japanese living in Korea during the period from 1908 to 1929. The percentage for the Japanese was 9, while that for the Koreans was 7.9. A statistical study of the various stages of syphilis in both races, however, shows considerable variations. The first stage of syphilis is seen most commonly among Koreans of the upper class and to a less extent among Koreans of the lower class, while the Japanese in Korea showed the lowest percentage. The second and third stages of syphilis are more prevalent among Koreans of the lower class, while latent and hereditary syphilis occur more frequently among Japanese. The authors are inclined to believe that the strain of spirochetes in the Koreans is not influenced so much by modern treatment for syphilis as the strain in the Japanese. The question of the existence of a relationship between the incidence of dementia paralytica in a race and the prevalence of malaria in the country that race inhabits is answered affirmatively. The authors state that Korea is still malaria-infested, and because the natives take little precaution against malaria the occurrence of this disease among Koreans is overwhelmingly greater than among Japanese living in Korea. They also cite statistics to show that there

was a negative reaction to malarial inoculation in 9.3 per cent of Japanese suffering from dementia paralytica, while the percentage of negative reactions among Koreans was 52.9. Spontaneous cessation of paroxysms was observed among Japanese in 16 per cent of the patients and among Koreans in 35 per cent. The authors conclude that the majority of Koreans pass through an attack of malaria before adolescence and thus secure immunity to the disease, and with it a possible resistance to metasyphilitic developments. This theory, however, is offered tentatively only.

NOTKIN, Poughkeepsie, N. Y.

HISTOLOGIC LESIONS OF THE CENTRAL NERVOUS SYSTEM IN EXPERIMENTAL INTOXICATION PRODUCED WITH HYDRAZINE HYDRATE. GIUSEPPE CURTI, Riv. di pat. nerv. **42**:27 (July-Aug.) 1933.

Study of the histologic changes of the brain following intoxication with different types of amines, histamine, bulbocapnine, indole, nicotine and mescaline, is attracting more and more the attention of neuropsychiatrists for the possible correlation of these changes with the histologic changes that have been reported in some types of mental disease. The author used hydrazine hydrate, a typical amine (NH_2-NH_2OH), which he injected subcutaneously into rabbits in a dose of 1½ grains (0.97 Gm.) in 5 cc. of water per kilogram of body weight. The symptoms following the injection were similar to those mentioned in intoxication due to ammonia, i. e., irritability, convulsive manifestations, coma, increase in salivation and vomitus and, in the dog, a tendency to catalepsy. Death generally followed severe convulsive manifestations and occurred a few hours after the administration of toxic doses.

The main histologic lesion of the nerve cells is the process of vacuolation, which is associated with perivascular edema. In some nerve cells there is only one vacuole, whereas in others many more are formed. The cells are more damaged in animals which have had repeated doses of the toxic substances. The nerve fibers are generally better preserved, but swelling of the myelin sheaths is found here and there, particularly in the pons. In some instances the fusion of swollen myelin sheaths leads to the gradual formation of the so-called grapelike areas of disintegration described by Buscaino. The astrocytes disclose slight degenerative changes, with slight clasmatodendrosis. The oligodendroglia show acute swelling, particularly in the gray matter. The microglia appear generally well preserved, and only here and there seem to undergo acute swelling.

The author compares the vacuolar degeneration of the nerve cells with the changes reported in experimental intoxication with histamine hydrochloride (Roberti, Buscaino), bulbocapnine (Pero, Hechst), indole and histamine (Ferraro and Killman).

FERRARO, New York.

THE INTIMATE RELATION BETWEEN OPHTHALMOLOGY AND NEUROLOGY. BERNARD SACHS, Arch. Ophth. **10**:433 (Oct.) 1933.

The author, in considering some reminiscences, believes that a close relationship is essential between the various specialties of medicine. He believes that the ophthalmologist should insist that every medical student graduated have a fair knowledge of the diseases of the eye. Not only is this of importance in such conditions as arteriosclerotic changes in the eye, but it is of special importance when one considers the differential diagnosis of optic neuritis and of choked disk. The condition of the pupils from the standpoint of the general physician and of the neurologist is much more important than any changes in the fundi which may be present in respect to the diagnosis of general diseases and of diseases of the central nervous system. Further, not merely is the loss of reaction to light and of prompt contraction during accommodation significant of tabes dorsalis, but the sluggish reaction both to light and during accommodation in all forms of constitutional syphilis is one of the most useful symptoms that a patient can present.

Sachs, speaking as a neurologist, states: "Where I find pupils mobile to light and sluggish during accommodation and where I find, in addition, an irregular contour of the pupil when the pupil responds or fails to respond to light, I shall risk the diagnosis of constitutional syphilis whether all possible reactions are corroborative of that diagnosis or not." He has found repeatedly that the ophthalmologist does not seem to be as conscious of the tremendous diagnostic importance of the pupillary reaction as the neurologist. The matter of fine discrimination between functional and organic disturbances of the eyes has been mentioned time and time again, especially in medicolegal cases. Sachs states that there is no more difficult task than the one of convincing a jury or a judge of the existence of purely functional amblyopia following trauma.

The author closes the article by begging that the ophthalmologist maintain the closest possible relation with neurology, with otology and, even more, with general medicine.

SPAETH, Philadelphia.

SPASMODIC TORTICOLLIS IN AN OLD ENCEPHALITIC WITH CERVICAL LESIONS. A. RAYBAUD, Rev. d'oto-neuro-opht. **11**:185 (March) 1933.

The question whether the pathogeny of spasmodic torticollis is purely mental or whether there is an organic substratum localized in the striate nucleus and allied to an encephalitis is still an open one. The following observation supports the latter view. A woman, aged 40, had always enjoyed perfect health, except for some attacks of rheumatism. In 1922 and in 1928, periods of somnolence had been noted, the latter attack being accompanied by diplopia. In 1930, pain in the cervical region, accompanied by spasms of the muscles of the neck, occurred. During the ensuing two years these crises of torticollis persisted and grew worse, and the head became fixed in the position of torsion toward the right and flexion on the right shoulder. Eventually a deformity appeared in the cervicodorsal region, and a cervical rib was discovered on each side. Improvement set in, the spasms ceased, and voluntary movements of the head became possible. Numerous examinations revealed integrity of the pyramidal system (except a slightly exaggerated patellar reflex on the left side), the sensory system and the organs of special sense. Lateralization of the extended arms and past pointing to the right were noted on several occasions. Roentgenograms of the cervical spine in 1930 revealed areas of decalcification and a suggestion of osteophytosis affecting the left transverse processes. In 1932, marked spondylophytosis of all the cervical vertebrae was revealed. The reaction to the Bordet-Wassermann test was negative.

The vestibular signs are important in the retrospective diagnosis of epidemic encephalitis and in their rôle of determining spasmodic phenomena, as suggested by Barré. The relation between the cervical lesions and the encephalitis, on the one hand, and the spasmodic torticollis, on the other, is that of cause and effect. The spasmodic torticollis here is an extrapyramidal reflex, set in motion and maintained by a cervical irritation and resulting in a cervical, spasmodic and hypertonic motor reaction.

DENNIS, Colorado Springs, Colo.

TREATMENT OF NEUROSYPHILIS: REVIEW OF RESULTS IN SIX HUNDRED AND EIGHTY PATIENTS. H. HANFORD HOPKINS, Arch. Int. Med. **52**:66 (July) 1933.

This article is a study of the effects of various methods of treating neurosyphilis in the different forms of the disease. Six classifications are reported: (1) early neurosyphilis; (2) diffuse late neurosyphilis; (3) dementia paralytica; (4) tabes dorsalis; (5) optic atrophy; (6) vascular neurosyphilis. Seventy-five per cent of the patients in the first group (early neurosyphilis) gave a history of a chancre; only about 50 per cent of the others remembered the primary stage. Types of treatment were classed as: (1) poor treatment, i. e., less than one year; (2) routine treatment, i. e., an arsphenamine product, iodides and mercury or bismuth administered repeatedly for more than a year; (3) intensified treatment

—courses of from ten to fifteen injections of old arsphenamine, with mercury, bismuth and iodides; (4) intradural treatment, i. e., routine treatment together with injections given according to the Swift-Ellis method; (5) tryparsamide in addition to routine treatment; (6) malaria in addition to routine treatment.

In the cases of early neurosyphilis, intensive routine treatment invariably gave excellent results. Favorable results were reported in most instances of even inadequate treatment in this group. In diffuse late neurosyphilis, the intradural and tryparsamide technics were much more effective than the intensive routine, and malaria was effective in more than half of the patients in this class who were subjected to it. In the group of tabes and dementia paralytica, malaria was the most successful of the methods, giving favorable results in 65 per cent of the patients who received it. In optic atrophy, the Swift-Ellis technic was successful in several cases in which routine treatment had proved inadequate. The correlation between clinical and serologic improvement was roughly proportional to the duration and type of pathologic involvement.

DAVIDSON, Newark, N. J.

TRAUMATIC ENOPHTHALMOS FROM NERVE LESIONS. F. FARNARIER, Rev. d'oto-neuro-opht. **11**:177 (March) 1933.

Many different conditions, having in common only retraction of the globe in the orbit, have been embraced under the term traumatic enophthalmos. A man, aged 20, was struck on the left side of the face, causing a wound of the soft tissues and fracture of the edge and floor of the orbit that extended into the maxillary sinus. Four months later there were still intense photophobia, pain in the globe, cheek, lower lid and head and, intermittently, fog before the left eye. There existed also marked enophthalmos without deviation, narrowing of the palpebral slit, increased by an effort to open the eyes widely, and mydriasis of the left pupil which had normal reactions. The eyegrounds were normal; visual acuity was good; the visual field was slightly contracted concentrically, and there was no trace of diplopia. Hypesthesia of the left cornea, the lower lid and the infra-orbital region was marked. Ocular pain was produced by pressure, but especially by light.

Only an involvement of the numerous nerves in the orbit could explain the enophthalmos and the pupillary and sensory troubles. A lesion of the ophthalmic ganglion could cause the enophthalmos and insensitivity of the cornea. The absence of myosis is due to the fact that the lesion did not involve the dilator fibers, which do not go through the ganglion but by way of the long ciliary nerves from the nasal branch. The mydriasis was probably caused by incomplete paralysis of the motor root of the ophthalmic ganglion; photophobia by lesion of the ciliary nerves or of the small root of the ganglion; the sensation of fog by a circulatory disturbance, and the narrowing of the visual field by the enophthalmos. The actual lesion was probably a hemorrhage located behind the ophthalmic ganglion, which involved the sympathetic and sensory roots and perhaps also the motor root, as well as the three branches of the ophthalmic nerve.

DENNIS, Colorado Springs, Colo.

SECOND ATTACKS OF POLIOMYELITIS. T. B. QUIGLEY, J. A. M. A. **102**:752 (March 10) 1934.

Quigley reviews the literature on secondary attacks of poliomyelitis and presents a similar case in which the changes found at necropsy were typical of those observed in the eighty-one fatal cases of poliomyelitis of the 1931 epidemic studied previously in the author's laboratory, of which 75 per cent showed a similar mild hemorrhagic gastritis and 86.7 per cent definite hyperplasia and congestion of the lymphoid tissue. While the greater part of this hyperplasia is undoubtedly a manifestation of the disease, the fact that in more than 30 per cent of the series the faucial tonsils had been removed suggests some degree of constitutional hyperplasia in certain cases, at least. Although microscopic examination disclosed

definite lymphoid hyperplasia in the thymus, the organ was somewhat smaller than is usually found in poliomyelitis. The average weight in the disease is almost always from 10 to 15 per cent greater than the average normal weight. The pale, muddy, granular appearance of the cut surface of the liver, which defies accurate description, is believed to be almost peculiar to the disease. It is quite distinct from the diffuse cloudy swelling encountered in most infectious diseases but has no specific microscopic characteristics. On several occasions, when clinical opinions were at variance, a diagnosis has been ventured on the appearance of the liver alone, and it was later confirmed by examination of the central nervous system. The changes in the central nervous system in the author's case illustrate the often observed lack of correlation between clinical and pathologic observations in acute poliomyelitis. No involvement of the extremities was apparent in the second attack, yet marked degeneration of the nerve cells, neuronophagia, congestion and edema were observed at every level of the cord examined post mortem.

EDITOR'S ABSTRACT.

CEREBRAL MOTOR CORTEX OF THE PORCUPINE. ORTHELLO R. LANGWORTHY and CURT P. RICHTER, J. f. Psychol. u. Neurol. **45**:138, 1933.

The authors studied the nervous system of the porcupine, paying special attention to the neural mechanism subserving the erection of the quills. They stimulated the cerebral cortex with a faradic current, using a stigmatic electrode mounted on a delicate spring to delimit the electrically responsive motor area. The large indifferent electrode was attached to the animal's back. Eleven porcupines were subjected to this investigation. The animals were anesthetized with ether before the experiment, and the anesthesia was continued throughout the entire experiment. The cortex was exposed on both sides so that the entire dorsal surface could be explored. It was found that the electrically responsive areas are well differentiated and that centers may be localized which respond to stimulation with contraction of the muscles of the face, forelimb, hindlimb and tail, all on the contralateral side. There are two areas representing the tail. Application of the electrode to one of these produced a movement of the tail to the opposite side and downward, whereas application of the electrode to the other area caused extension of the tail in the midline. The tail is used for defense and is the only part of the body that can be moved speedily and with precision. The panniculus carnosus is also represented in the motor cortex; stimulation of this area caused a turning of the body toward the opposite side. The extension of the tail in the midline is also dependent on contraction of the panniculus carnosus. No elevation of the quills occurred on stimulation of the cerebral cortex, although contraction of the panniculus carnosus caused a tightening of the skin over the back, which probably contributes to the effective functioning of the quill apparatus.

KESCHNER, New York.

STUDIES ON CEREBROSPINAL FLUID: II. THE OCCURRENCE OF LEAD IN CEREBROSPINAL FLUID. I. M. RABINOWITCH, A. DINGWALL and F. H. MACKAY, J. Biol. Chem. **103**:725 (Dec.) 1933.

After chemical and spectrographic analysis of cerebrospinal fluid for lead in a variety of conditions, the authors report the following results: 1. Lead was found in the cerebrospinal fluid of every person known to be suffering from lead poisoning. 2. Lead was found in the fluid of a lead worker with no clinical signs of lead poisoning. 3. Lead was also found in the fluid in isolated persons having no histories of undue exposure to the metal. In these cases, however, with one exception, the amounts were very small when compared with those found in persons with lead poisoning.

An attempt was made to induce the appearance of lead in the cerebrospinal fluid by producing acidosis. Acidosis was obtained by the administration of ammonium chloride, and the degree of acidosis was determined by the carbon dioxide-combining

power of the plasma. Whether lead was or was not liberated from the body was determined by the amount of lead in the urine during acidosis compared with that found before acidosis. Of twenty persons suffering from a variety of neurologic conditions other than lead poisoning, in whom no lead was found in the cerebrospinal fluid before acidosis, five showed lead in the cerebrospinal fluid during acidosis. As before acidosis, however, the amounts found were very small—approximately 10^{-4} mg. in 6 cc. of cerebrospinal fluid.

The data cited lead to the conclusion that when the precautions which are described for the detection of lead in cerebrospinal fluid are strictly adhered to, lead is found in appreciable quantities (more than 10^{-4} mg. in 6 cc. of fluid) in lead poisoning only.

DAILEY, Boston.

THE IMPORTANCE OF INTRODUCING PSYCHIATRY INTO THE GENERAL INTERNSHIP. F. G. EBAUGH, J. A. M. A. **102**:982 (March 31) 1934.

Ebaugh believes that psychiatry should be represented in the general internship as part of the basic preparation for the practice of medicine. Such participation of psychiatry in straight, rotating and mixed internships is now possible in most hospitals through: (1) combined clerkships and internships and service in state hospitals during the summer months, (2) active psychiatric consultation service in both ward and outpatient departments, (3) liaison work between the various departments, especially with medicine and pediatrics, and (4) the allotment of definite time for psychiatry, preferably from two to four months in rotating schedules. There should be greater utilization of the well equipped psychopathic and state hospitals, whereby affiliated schedules can be developed for intern training. At the present time less than one fourth of the 7,357 interns now in training have the benefit of psychiatric training. In general, these psychiatric services are well appreciated by the intern and accepted by the staff and administrators of these hospitals as an essential part of the basic training. Psychiatric training in the future should be considered as a requirement for licensure. This will be a great educational step forward in protecting the public from the quack and further emphasizing the importance of the psychologic aspect of all medicine as well as furthering greater cohesion and correlation between the various fields of medicine. Introducing psychiatry through the general internship will be a great impetus to the changes now taking place in undergraduate and graduate psychiatric teaching schedules. It should play a rôle in breaking down the tendency for immature specialization in various fields and in the acceptance of psychiatry as a fundamental phase of all medicine.

EDITOR'S ABSTRACT.

FURTHER EXPERIMENTS UPON THE EXTIRPATION OF MAUTHNER'S NEURONES IN AMPHIBIAN EMBRYOS (AMBLYSTOMA MEXICANUM). S. R. DETWILER, J. Exper. Zool. **64**:415 (Feb. 5) 1933.

Axolotl larvae (Amblystoma mexicanum) and the larvae of Amblystoma punctatum lacking one Mauthner neuron exhibit similar behavior. On prolonged tactile stimulation the larvae exhibit jerky, poorly coordinated, swimming reflexes which they are incapable of sustaining, and exhaustion ensues much more quickly than in normal animals. Larvae lacking both Mauthner neurons do not show any greater functional disturbances. There was no evidence of postural defects or disturbed equilibrium during swimming. When both the ear and Mauthner's neuron are excised on the same side, there is evidence to show that the sustained motor activities are slightly more impaired than when the Mauthner neuron alone is excised. When both ears are excised, but with Mauthner's cells intact, the larvae show extreme equilibratory disturbances with lack of precise movements. They exhaust as readily as do larvae lacking Mauthner's neurons. The failure to continue prolonged muscular activity appears to be associated with the greatly lowered muscle tonus resulting from the absence of the ears.

The results bear out former conclusions that the Mauthner neurons are not indispensable for maintaining equilibrium, and that they play an important rôle

in transmitting impulses which are essential for prolonged coordinated motor activities. That they may be concerned with speed and precision is also suggested, since in their absence there is evidence that the larvae are not so capable of exhibiting sudden lurching movements.

WYMAN, Boston.

THE THYROID GLAND IN MENTAL DEFICIENCY: A HISTOLOGICAL STUDY. J. L. NEWMAN, J. Ment. Sc. **79**:464, 1933.

Newman reports a histologic study of the thyroid glands of ninety mentally deficient children between the ages of 1 and 19, who died at the Fountain Mental Hospital in England. In clinical analysis of the cases he uses Tredgold's classification of primary and secondary mental deficiency. By primary mental defect he means those conditions caused by some anomaly or defect of the germ plasm, while secondary mental deficiency is due to traumatic, toxic and infectious agents. Sixty-five cases were in the primary group. The author gives a thorough analysis of the cases from the point of view of the histologic picture, clinical aspects, age at death, state of nutrition and diet. He emphasizes the extraordinary variations in the histologic picture in the so-called normal thyroids, and finds the same variation in the thyroids of mentally deficient children. No consistent findings were established correlating mental deficiency with a specific histologic abnormality of the thyroid gland. He conservatively remarks that there is evidence of qualitative and quantitative lack of parenchyma, suggesting a diminution of the function of the thyroid gland. The thyroid glands of children usually show a hyperplasia which is not seen in adults. The state of nutrition has no connection with the histologic appearance of the thyroid gland. There was no correlation between the thyroid and the thymus glands. The review of the subject occupies forty pages and is done with great care and thoroughness.

KASANIN, Howard, R. I.

AN OPERATION FOR A SUPRASELLAR TUMOR. C. VINCENT, R. ONFRAY and MATTE, Ann. d'ocul. **170**:79 (Jan.) 1933.

Vincent, Onfray and Matte present the case of a highly myopic patient who was under observation for visual disturbance and for symptoms of narcolepsy. Examination of the eyes showed temporal hemianopia on the left and hemiachromatopia on the right side. Vision with correction was: left eye, 1/15, right eye, 2/10. A roentgenogram of the sella turcica showed no enlargement of it but revealed involvement of the posterior clinoid processes. Neurologic examination confirmed the presence of a suprasellar tumor. During operation a soft, vascular suprasellar meningioma, the size of a large tangerine, was found extending under both hemispheres. It was removed by means of an aspirator, with electrocoagulation and careful hemostasis. On the following day there was improvement in visual acuity, and the psychic disturbances had disappeared. The visual fields were about normal and vision was 4/10 in the right and 6/10 in the left eye. Dupuy Dutemps stated that although surgical intervention is indicated in suprasellar tumor, one must remember that radiotherapy is efficacious in tumors of the sella turcica. Morax believed that in cases of the type of this one only surgical treatment could produce permanent results. It is interesting to note the rapidity of functional improvement, which leads to the belief that there is no lesion of the optic fibers, and to the supposition that compression acts not on the fibers themselves but on the blood supply of the chiasm.

BERENS, New York.

METASTATIC CARCINOMA OF THE EYE AND BRAIN. ISIDORE FINKELMAN and LEO L. MAYER, Arch. Ophth. **10**:83 (July) 1933.

Since the first report by Peria in 1872 and up to 1923, only 130 cases of carcinoma metastasizing in the eye were to be found reported in the literature. In 1923, Usher reviewed all the cases reported up to that time and added 3 of his

own. In 1932, Michàil reviewed the literature since Usher's paper and found 129 additional cases. Four others have been reported since. It is of interest that Finkelman and Mayer were first aroused by the fact that the right pupil, in the case reported, remained responsive to light despite the fact that the patient was totally blind in that eye. It was thought that perhaps a basis for this interesting phenomenon could be found, but the enormous involvement of the brain made any lesion simply one of many as far as localization was concerned. The tumor was a medullary carcinoma, identical with a primary neoplasm in the breast, and was first diagnosed in the eye two years after a lump was first discovered in the right breast. At the time it was discovered in the eye, the carcinoma had advanced in the breast to such a degree that it was considered inoperable. In the same case metastases were found in various portions of the skeleton and the dura mater, the pia-arachnoid, the left temporal lobe, the left parietal region, the left occipital lobe, small metastases in the pons and cerebellum and many small nodules scattered throughout the white matter of the right hemisphere.

SPAETH, Philadelphia.

PAIN. MAURICE DELAET, J. belge de neurol. et de psychiat. **33**:165 (Feb.) 1933.

DeLaet tried to determine the significance in the basal metabolic reactions provoked by pain from the point of view of arterial pressure and of changes in the psychogalvanic reflexes. He also studied changes in the p_H value of the urine by determining the reaction just before and after a painful experience. For his experiment he selected patients, most of whom were to have some oral operation done, and divided them into two groups, those who apparently had no pain and those who manifested pain. The patients who gave no evidence of pain had had local anesthesia, and the patients in whom pain was manifested showed a well defined fall in the p_H value of the urine. In two instances there was a slight elevation of the p_H value and these manifested no reaction after several intradermal injections of 25 cc. of horse serum. Apparently, local anesthesia abolished the tendency to acidification, since in none of this group was the p_H lowered. In one case there was no change under anesthesia, but when the same procedure was carried out in this patient without anesthesia, and with subsequent violent pain, there was a fall of 0.7 in the p_H value of the urine. In a comparative study of the specific gravity it was noted that the fluctuations of the p_H could not be correlated with those of the specific gravity. It is assumed from these data that pain of more than a certain degree provokes in most persons a sympathicotonic reaction. On the other hand, it was noted that emotion had a tendency to alkalinize the urine.

WAGGONER, Ann Arbor, Mich.

SOME CONSIDERATIONS ON THE PATHOGENESIS OF DIABETES INSIPIDUS. ALBERT SALMON, Schweiz. Arch. f. Neurol. u. Psychiat. **33**:95, 1933.

Diabetes insipidus is generally transitory following experimental injury of the tuber cinereum and may be lacking altogether when the entire floor of the third ventricle has been destroyed by disease. There are no constant relations, furthermore, between polyuria and either hypofunction or hyperfunction of the anterior lobe of the hypophysis. Salmon seeks, with the aid of numerous citations from the literature, to account for these discrepancies. Normal urinary secretion, he assumes, is dependent on the existence of a proper balance between the respective functional states of the sympathetic and parasympathetic systems. In the presence of a lesion sufficiently extensive to destroy the hypothalamic centers of both systems, diabetes insipidus would, according to this hypothesis, be lacking. The vegetative equilibrium is further assumed to be related to a "hormonal equilibrium." After citing instances of an antagonism between the respective actions of solution of pituitary and epinephrine, the author points out that the latter, when given in small doses, may act as a vasodilator and diuretic, particularly in vagotonic states or states of depressed sympathetic activity. Injections of epinephrine often aggra-

vate a preexisting polyuria, and autopsy has revealed, in cases in which the polyuria ceased in the terminal stages, changes in the suprarenal glands as well as in the hypophysis.

DANIELS, Denver.

AGE INCIDENCE OF PARESIS. MAGNUS C. PETERSEN, Am. J. Psychiat. **12**:521 (Nov.) 1932.

Dementia paralytica in the aged, presumed by most authorities to be rare, is found by Petersen's statistical study to be not at all infrequent. The incidence among males per hundred thousand of the population was 40 between the ages of 55 and 65 years, 23 in the ages from 65 to 75, and 10 per hundred thousand in the group of men over the age of 75. Among women, dementia paralytica is less common, except in the younger groups. From 25 to 30 years of age, the rate per hundred thousand was 8 for males and 13 for females. In all older classes, however, the men outnumbered the women considerably. The incidence of dementia paralytica in the hospital for mental diseases studied was 8.2 per cent. Only cases confirmed by the examination of the spinal fluid were included in this calculation. The age range was from 22 to 77. The Wassermann test of the blood was positive in 82.5 per cent of the patients with dementia paralytica, but in the group of patients over the age of 65 positive Wassermann tests of the blood were found in only 70 per cent. Petersen believes that this accounts for the fact that dementia paralytica in the aged is so often diagnosed as arteriosclerotic or senile insanity. Examination of the spinal fluid, which affords the only certain means of differentiation, is not performed when there are no changes in the blood.

DAVIDSON, Newark, N. J.

A STUDY OF THE SEXUAL LIFE IN PSYCHOSES ASSOCIATED WITH CHILDBIRTH. E. W. ANDERSON, J. Ment. Sc. **79**:137, 1933.

Zilboorg's researches in psychopathology of the puerperal psychoses are reviewed by Anderson. He questions the finding of frigidity as a consistent phenomenon in these cases. Zilboorg also stated that a great majority of the patients had married after prolonged courtships and usually in the latter part of the second decade of life. The study was made of two groups of cases. In one group there were fifty young women in whom psychoses developed in connection with childbirth or pregnancy; this group is compared with another group of fifty young women in whom pregnancy or childbirth was not the precipitating factor. Both groups showed relative predominance of affective disorders. Marital unhappiness was more frequent in the control group, although in both groups the majority of patients claimed fairly satisfactory marriages. Auto-eroticism was found in only three cases in both groups. No material difference was revealed in the pre-psychotic sexual life in the two groups, and persistent frigidity could not be crystallized as a constant feature of the sexual constitution of the puerperal group. Finally, Anderson comes to the conclusion that there is no evidence that the psychoses associated with childbirth are necessarily due to the unsatisfactory sex life of the patients, with the puerperium acting as a specific precipitating factor.

KASANIN, Howard, R. I.

FAMILIAL OPTIC ATROPHY. FAVORY and PÉTRIGNANI, Rev. d'oto-neuro-opht. **10**:494 (July-Aug.) 1932.

This observation concerns four brothers aged 29, 27, 24 and 11 years. The three eldest had identical histories: vision was excellent up to the age of 11 years, when it failed rapidly. The family history was without significance, the parents, grandparents and collaterals having enjoyed good health and vision. Examination revealed vision of 1/50, and visual fields with normal peripheral limits but with large central scotomas; horizontal nystagmus was present and the chromatic sense was absent in two of the patients. The fundi exhibited lesions of disseminated chorioretinitis. General, neurologic and roentgenologic examinations gave negative

results. The Wassermann reaction was negative in all four patients and in the parents. The youngest, who had been in good health and had had excellent vision, shortly before observation had suffered from delirious excitement, followed by amnesia, which was probably of epileptic origin.

The hereditary character of Leber's disease is not apparent in this family, but it is believed that consanguinity in the parents of the boys (they were second cousins) has brought to light a hereditary blemish that was latent in the ancestors. As is frequently the case, no etiologic factor has been discovered. Endocrine disturbances were especially looked for but were not found.

DENNIS, Colorado Springs.

BEHAVIOR DIFFICULTIES THAT BAFFLE TEACHERS. VIRGIL E. DICKSON, J. Juvenile Research **16**:93 (April) 1932.

In classifying their problems of discipline according to gravity, teachers are inclined to emphasize as serious those which interfere with the pedagogic routine, such as whispering, disobedience and meddlesomeness. They attach little significance to such abnormalities as shyness, fear or day-dreaming. Mental hygienists, on the other hand, reverse this order, considering that those problems which cause most trouble to the teachers are evidences of a strengthening personality trying to express itself. The traits, such as cruelty, sensitiveness and shyness, which cause little trouble in school, are considered more serious by psychiatrists and psychologists. In a statistical study, Dickson finds that misbehavior is commoner among boys and is found at all ages and in all grades, with a central tendency in the fifth grade and at the age of 11, and that these problems are unrelated to intelligence but are to a great extent associated with physical difficulties. Most common of these is endocrinopathy. In practically all cases, there was misunderstanding or mismanagement at home. The teacher should be trained to recognize these problems as well as to evaluate them. The more serious cases should be referred to a psychiatrist. Unfortunately, the author does not tell the psychiatrist what to do with these children.

DAVIDSON, Newark, N. J.

THE COURSE OF HENRY'S REACTION IN EXPERIMENTAL MALARIA. R. LEROY, V. CHORINE and G. MEDAKOVITCH, Encéphale **28**:372 (May) 1933.

The technic of the so-called Henry reaction is described. This involves a series of dilutions of serum to be tested with melanin and iron albuminate. The uses and values of the tests are summarized as follows: 1. The melanin reaction in malaria is positive on the third day of the infection, increasing in intensity until it reaches its maximum at about the sixth or seventh paroxysm. As quinine is given, the reaction remains stationary for about twelve days, then steadily diminishes in intensity, to disappear from thirty to fifty days after the beginning of the treatment with quinine. 2. The ferrous reaction becomes positive after the third or fourth attack, and increases rapidly until the seventh or eighth. Its subsequent course is essentially similar to the melanin reaction. 3. Henry's reaction is negative at the onset of the paroxysm. 4. The serums of malarial patients flocculate in distilled water after the fourth or fifth attack. 5. There is perfect correlation between the clinical findings and the evolution of these tests: if they remain positive more than fifty days, the patient is not cured. 6. The substance responsible is thermolabile.

ANDERSON, Los Angeles.

ACTIVATION OF THE MESENCHYME WITH THERAPEUTIC MALARIA. WALTER L. BRUETSCH, J. Nerv. & Ment. Dis. **76**:209 (Sept.) 1932.

The observations of changes in mesodermal tissue were made by the author on patients who had died during induced malaria. The endothelial cells of the larger vessels appeared less stimulated to growth. Many undifferentiated mesenchymal cells were found newly formed in the connective tissues of the organs. A cell called

the histiocyte, normally present in the liver and spleen, and actively phagocytic, was considered by the author to represent the type most important in the therapeutic action of malaria. Perivascular collections of histiocytes are found in the subcortical white matter. No phagocytic action on the part of the microglia was found. Stimulation of the monocytes, myelocytes and erythrocytes, as well as of the undifferentiated mesenchymal cells of embryonic character, was found to be considerable. It is the author's opinion that the resting histiocyte of the splenic pulp was activated to a higher degree of phagocytosis than in any other disease examined.

HART, Greenwich, Conn.

THE LIVER TREATMENT OF FUNICULAR SPINAL DISEASE. H. DEML, Arch. f. Psychiat. **99**:57 (Feb.) 1933.

Twenty-eight cases of pernicious anemia in which involvements of the spinal cord occurred were observed for several years. In twenty-three of these liver therapy (raw liver and hepartrat) was used. In eight patients there was improvement; in four the condition remained unchanged; eleven died (five died from intercurrent infections). In fourteen of the twenty-eight patients paresthesias in the extremities were the first symptoms; in twenty-five there was a pronounced disturbance of deep sensation, and in nineteen there were disturbances of superficial sensation. In five patients there were purely spastic pictures, and in seven the conditions were those of pseudotabes. Only thirteen patients showed typical forms of combined system disease. Nystagmus was found in four patients, intention tremor in three and schizophrenia-like mental disturbances in two. Deml concludes that liver therapy is indicated in pernicious anemia with neurologic disturbances, and that although it is not infallible a large number of cases can be influenced by this treatment.

MALAMUD, Iowa City.

NEUROSPONGIOBLASTOMA, A PRIMARY TUMOR OF THE BRAIN IN DISSEMINATED NEUROSPONGIOBLASTOSIS (TUBEROUS SCLEROSIS). J. H. GLOBUS, I. STRAUSS and H. SELINSKY, Ztschr. f. d. ges. Neurol. u. Psychiat. **140**:1 (June) 1932.

Globus, Strauss and Selinsky report eleven cases of tumor of the brain in fully developed or abortive tuberous sclerosis. These tumors are characterized by localization in the striothalamic region, by the typical cellular arrangement in nests, by the predominance of spongioblasts in the tumor and by the presence of tuberous nodules in the brain. Since both spongioblasts and neuroblasts are found in varying numbers in the tumors and the nodules, it is suggested that the term "neurospongioblastoma disseminata" be used to describe such growths, especially when they are associated with tuberous sclerosis. The cases support Bielschowsky's hypothesis that tuberous sclerosis is caused by a blastomatous tendency on the part of the glial elements. Globus, Strauss and Selinsky would broaden this hypothesis to include the nerve cells.

ALPERS, Philadelphia.

BLOOD CHOLESTEROL STUDIES IN MENTAL DISEASE. PURCELL G. SCHUBE, Am. J. Psychiat. **12**:355 (Sept.) 1932.

On admission of patients to the wards or clinic of the Colorado Psychopathic Hospital, 5 cc. of blood is drawn by venipuncture. Evaluation of the cholesterol content was made in the specimens drawn in 200 neuropsychiatric cases. As a control, a corresponding study was made of blood drawn from 54 normal volunteers who presented no clinical evidence of neurologic or psychiatric disturbance. Duplicate estimates were made on each specimen and the average of the two findings was taken as the true content. Cholesterol was estimated by the Myers-Wardell method. The normal persons showed a range of from 110 to 195 mg. per hundred cubic centimeters of whole blood, while the neuropsychiatric group exhibited a much greater scattering of observations, from 30 to 350. The average value was

148 in the normal and 129 in the not-normal groups. The observations in the not-normal group were not classified as to disease or personality type, nor were any clinical conclusions drawn from the results. DAVIDSON, Newark, N. J.

RESEARCHES ON THE CONTRACTURE OF SKELETAL MUSCLE. FREDERIC BREMER, J. Physiol. **76**:65 (Sept. 16) 1932.

Myotonic rigidity of human pathology presents striking analogies in properties and mechanism with neuromuscular contracture in Anura. Contracture by excitation of skeletal muscle seems to be a primitive form of reaction of the muscle fiber, connected with an "excitable substance" of long chronaxia and characterized by the slowness of development and dissipation of its excitatory process, its absence of wavelike propagations and its persistence throughout the duration of a galvanic stimulus (absence of adaptation). A comparative study of the same muscle, such as the gastrocnemius in various Anura, suggests that the tendency of a muscle to contract depends on its functional specialization, in the sense that habitual participation of the muscle in a rapid alternating movement (swimming and jumping) accelerates its evolution toward a stage characterized by a lack of or by the presence of only a feeble aptitude for contracture by excitation.

ALPERS, Philadelphia.

TWO METHODS OF INVESTIGATING DISEASE OF THE CORPUS STRIATUM. C. J. MUNCH-PETERSEN and T. B. WERNØE, Am. J. M. Sc. **184**:407 (Sept.) 1932.

Two different methods which are thought to indicate striatal disease are compared. The epinephrine-liquor saccharum test is supposed to be followed by a rise in the sugar content of the spinal fluid as recovered on lumbar puncture. A smaller rise than is expected is interpreted as a definite sign of a direct striatal condition. The bimanual faradic irritation test causes contraction of the muscles in the upper extremity, resulting in pronation, flexion of the wrist joint and flexion of the elbow. In striatal conditions this response may be absent or atypical. In an attempt to evaluate these methods, 33 cases were examined with both tests with an agreement of 67 per cent and a disagreement in 21 per cent, with no decision. It is supposed that the reaction to the epinephrine-liquor saccharum test is released by vegetative functions, while that to the bimanual faradic irritation test is released via motor centers.

SYPHILITIC HYDROCEPHALUS IN THE ADULT. J. G. GREENFIELD and R. O. STERN, Brain **55**:367 (Sept.) 1932.

Seven verified cases of syphilitic hydrocephalus in adults are reported in detail. Headache was usually the first symptom and was present in all the cases. Four cases presented attacks of loss of consciousness, sometimes of convulsive character. In every case palsies of the cranial nerves were prominent. Papilledema was present in every case except one and in four cases had progressed to optic atrophy. Weakness or sensory disturbances were not prominent, although there was tetraplegia in one case. The mechanism of hydrocephalus was difficult to ascertain; however, in four cases there was evidence of blocking of the foramina of exit from the fourth ventricle. Of histopathologic interest were the intensity of the neuroglial reaction around the medulla, on both its ependymal and its pial surfaces, and the frequency of giant cells in the granulation tissue.

MICHAELS, Boston.

Society Transactions

NEUROLOGICAL SOCIETY OF PARIS

Dec. 7, 1933

M. Clovis Vincent, *President*

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Abstracted by Walter Freeman, M.D., Washington, D. C.

Alterations of the Cerebellum and of the Spinal Ganglia in Friedreich's Disease. J. Lhermitte.

A girl presented rather typical signs of Friedreich's ataxia, with diminution in strength, loss of reflexes, diminution in deep sensibility and a positive plantar reflex. The upper limbs were involved, and there were kyphoscoliosis, tremor of the head, drooping of the eyelids, nystagmus and monotonous speech. The patient was irritable and required commitment. Pathologic examination revealed marked degeneration of the spinal cord in both the lateral and dorsal funiculi, of the type usually found. In the spinal ganglia there was diminution in the capsular cells, with marked lymphocyte and plasma cell infiltrations and reduction in nerve fibers. The cerebellar cortex showed few fibers and Purkinje cells, the basket fibers often enveloping clear spaces. The Purkinje dendrites often showed swellings and serpentine twistings. These findings are new proof that Friedreich's disease is associated with lesions of the cerebellum.

Palatal Myoclonus: Pathologic Examination. Georges Guillain, R. Thurel and I. Bertrand.

This case was previously presented from the clinical standpoint. The myoclonia involved not only the muscles of the palate and pharynx but those of the diaphragm, the lip and the eyes, the orbicularis palpebrarum and even the muscles of the neck and those of the upper and lower limbs. In spite of its extent, the myoclonia was practically limited to the left side. It was absent during complete repose and, depending on the posture of the patient, could be brought out with considerable force. The movements were constant in rhythm at 130 per minute and entirely synchronous.

Necropsy showed a few small lacunae in the white matter and putamen on each side. In the pons there were several of these, and there was an infarct destroying practically half of the right tegmentum and involving the upper end of the right superior cerebellar peduncle. The degenerations were striking; the left red nucleus was extremely pallid, the right median fillet practically destroyed, the right central tegmental fasciculus degenerated and the right olivary body greatly enlarged and pale. Areas of a similar type of hypertrophic degeneration were found in the left dentate nucleus. The median longitudinal fasciculus was unaffected.

The destruction of the central tegmental fasciculus at any point provokes degeneration as far down as the homolateral olive, and this is followed by pseudohypertrophic sclerosis of the olive and even sometimes by transsynaptic degeneration of the olivary cells and of the olivocerebellar fibers. Lesion of the red nucleus provokes retrograde degeneration of the superior cerebellar peduncle and occasionally severe atrophy of the corresponding cerebellar hemisphere and restiform body. Lesion of the dentate nucleus or of the restiform body causes a degen-

eration of the arcuate fibers and of the contralateral olivary body, with pallor of the fibrillary meshwork, but without transsynaptic degeneration of the central tegmental fasciculus. Degeneration of one olivary body is accompanied by degeneration of the olivocerebellar fibers and pallor of the cerebellar hemisphere of the opposite side and some atrophy of the opposite olive; in such cases the central tegmental fasciculus is entirely normal. This is not simple atrophy; it is accompanied by excessive proliferation of the neuroglia and even by hypertrophic degeneration of the olivary cells. It is probably a lesion of this type that conditions the myoclonic syndrome. The extension of the myoclonia from one side to the opposite side is apparently caused by progressive cellular degeneration. This hypothesis explains the slow development of the myoclonia after the original injury. There seems to be some relation between this type of olivary involvement and the tremors of pontocerebellar atrophy in the advanced stage. The olivary lesions as well as those elsewhere are similar. The one feature that seems constant in this condition is the olivary degeneration on the side opposite the myoclonia.

PARINAUD'S SYNDROME AND PALATAL MYOCLONUS: AN ANATOMIC STUDY. RAYMOND GARCIN, IVAN BERTRAND and PIERRE FRUMUSAN.

The anatomic foundation of conjugate paralysis of the eyes and of palatal myoclonia is incompletely understood; so a case combining the two is of unusual interest. The multiplicity of lesions in such cases makes the problem more difficult, since no one lesion can be blamed for the symptoms exhibited.

The patient was a woman, aged 65, who presented severe right hemiplegia with contracture and anarthria. There were spasmodic weeping and trouble in swallowing. The left side of the body was normal. Voluntary vertical movements of the eyes were practically abolished, and likewise the automatic vertical reflexes on lowering and raising the head. Convergence was abolished, but the lateral movements and the pupils were normal. The palate showed myoclonic twitchings at the rate of 150 per minute, sometimes skipping a beat, as in cardiac extrasystoles. The palatal reflex was abolished, but the pharyngeal reflex was preserved. The palate was partially paralyzed; nevertheless, there was no reflux of liquids through the nose. There were moderate hypertension and profound asthenia.

At autopsy the left hemisphere showed several small lacunae in the lenticular nucleus and thalamus; there was marked atrophy but no focal lesion in Wernicke's region; anterior to the left ganglion habenulae was a small softening in the wall of the third ventricle, just rostral to the posterior commissure. In the right hemisphere several small foci were found in the basal ganglia as well as a minute lesion in the knee of the internal capsule. Microscopically, it could be seen that many fibers of the posterior commissure were destroyed by the lesion, which also involved the capsule of the red nucleus and Meynert's fasciculus. The degeneration of the left pyramidal tract was profound; in the pons many foci were found ventrally, while the tegmentum was absolutely intact. The olivary bodies showed hypertrophic degeneration in the dorsomesial aspects, with overgrowth of neuroglia. The extraciliary fibers of both dentate nuclei were degenerated, but the hilus was preserved.

The Parinaud syndrome was believed to be due to the lesion in the vicinity of the habenula. The integrity of the central tegmental fasciculus is important, since in most of the cases of palatal myoclonus previously reported this structure has been incriminated. Severe degeneration of the dentate nuclei seemed to be of prime importance in this case, particularly since the dentate nucleus is tied up with the olivary bodies and the red nucleus in a functional triangle, as indicated by Guillain and Mollaret.

DISCUSSION

DR. DEREUX: The fact that a unilateral lesion could produce paralysis of associated vertical movements, and particularly that the lesion involved the posterior commissure, is of great importance and confirms the hypothesis previously expressed by Dr. Vincent and me.

DR. LHERMITTE: Pseudohypertrophic degeneration of the olives may occur without degeneration of the central tegmental fasciculus, and the fasciculus may degenerate without olivary hypertrophy; on the other hand, olivary pseudohypertrophy may reveal different features when studied minutely. Bielschowsky and Hirschfeld have reported olivary hypertrophy with cerebellar atrophy in dementia paralytica with marked vascular infiltration; on the other hand, the hypertrophy may be simple without inflammation. Thomas recently reported the occurrence of a cystic tumor of the pontile tegmentum in a patient who had presented myoclonia.

DEGENERATION OF THE CORPUS LUYSII AND ZONA INCERTA: CLINICOPATHOLOGIC STUDY. IVAN BERTRAND and RAYMOND GARCIN.

About fifteen anatomic studies of hemiballism have been reported, practically all showing a hemorrhagic lesion in the region of the corpus subthalamicum. The present case occurred in a woman, 61 years of age, who showed involuntary movements of the left arm and leg, which became progressively more tumultuous and did not cease even during sleep. Strapping the upper limb to the trunk had to be abandoned on account of the intense activity. The right side was normal.

Neurologic examination showed some weakness with spasticity on the left side, without painful phenomena, some disturbance of deep sensibility and weakness in ocular convergence. Synkineses of imitation, both homolateral and contralateral, were present, but no cervical reflexes. The movements gradually died away during hospitalization, but there occurred marked edema of the left hand and irritability, confusion and restlessness.

Necropsy revealed excellent preservation of the arteries and freedom from multiple softenings. There was rarefaction of the myelin sheaths, but no cell degeneration. The lenticular fasciculus was normal; the thalamic fasciculus was slightly affected; the fibers running to the external nucleus of the thalamus were degenerated, thus explaining the sensory disturbances. There was a peculiar fragility of the myelin in the zona incerta and below this in the corpus Luysii, without hemorrhage or softening, but resembling more the status precribrosus.

Thirteen previously reported cases have shown eight focal hemorrhages, four softenings and one tubercle. The attending mental disorders seem to be an integral part of the syndrome of involvement of the corpus Luysii. Moreover, the localized edema, probably due to vasomotor disturbance, is occasionally observed in certain thalamic lesions. The physiopathology of this condition is yet undetermined.

DISCUSSION

DR. LHERMITTE: Hemiballism is not always caused by destruction of the corpus Luysii. De Massary and I described a case showing lesions in the superior cerebellar peduncles and in the dentate nuclei. The edema is not that of the ordinary nephritic or cardiac type, but is apparently a nervous condition, provoking retention of chlorides; the fluid is not a simple saline solution but a dark amber, albuminous fluid, coagulating spontaneously, as I showed with Grenier in 1925.

DIFFUSE MELANOBLASTOMA OF THE NERVOUS SYSTEM. RAYMOND GARCIN, IVAN BERTRAND, ANDRÉ THÉVENARD and R. A. SCHWOB.

The diagnosis of primary melanoblastosis of the nervous system, of which about twenty cases have been reported, has never been made during life. The pathogenesis is even more difficult to settle, although the condition must arise in the chromatophores of the pia. These chromatophores have been found by Globus in a 5 month fetus.

In the case here reported there was extensive cerebral invasion following the vessels of the gray cortex, while sparing the white matter. The patient was an automobile mechanic, aged 31, who had jacksonian epileptic attacks every month with increasing violence and finally paresis of the right lower limb. He had had

a motorcycle accident without loss of consciousness. There was some inequality of the pupils; the spinal fluid showed 10 lymphocytes per cubic millimeter, with a rare mononuclear of normal character. Serologic reactions were negative. Mental disturbances and polyuria were present; the mental symptoms indicated a diffuse involvement, and there was no sign of intracranial hypertension, yet a cerebral tumor was suspected. Toward the end there developed painful hemiplegia requiring heavy doses of morphine and resulting in severe contractures and finally some nystagmus. The progressive painful manifestations led rapidly to death.

At necropsy the meninges were uniformly brown, as though from meningeal hemorrhage, yet there was no blood. The cortex and the pia were almost everywhere invaded by this process, and the thalamus was included to a lesser degree, while the basal ganglia were spared. Microscopic examination showed polyhedral cells extending along the vascular pathways in all directions; the cells were deeply pigmented with melanin. There was no secondary degeneration. "We may close by pointing out the eventual effect of repeated traumatism in the development of the effects we have described. Our patient, a tester in an automobile factory, during the whole preceding year had driven his machines over rough roads at an extremely rapid rate."

Systemic Opticocochleodentate Degeneration: A Study of the Familial Type. R. Nyssen and L. Van Bogaert.

Two children of the same family, otherwise normal, showed successively bilateral optic atrophy with blindness, bilateral deafness and mild spasmodic ataxia. There seems to have been degeneration of the primary optic neuron, of the primary and secondary cochlear neurons and of the dentate systems.

Tumor of the Fourth Ventricle; Spinal Hypertension Without Cranial Hypertension; Syndrome of the Inferior Triangle of the Floor of the Fourth Ventricle. J. A. Barré and L. Woringer.

A white man, aged 31, complained of dysphagia, a heavy tongue and slurring speech. He also suffered from painful tension in the neck, radiating to the mastoid region, and paresthesias of the finger-tips. These were insufficient to disable him, but he also suffered violent nervous spells, without loss of consciousness, that seemed to bystanders to presage immediate death. These began with hiccup for several hours and tenesmus, followed by weakness of the limbs with vertigo and falling, cyanosis, slowness and even arrest of pulse and respiration, and paralysis of the tongue. These phenomena, which might disappear in a few minutes, were first noted some ten years before the patient was seen. Examination showed the tongue deviated and hemiatrophic, the palate weakened and the mouth relatively motionless, but vestibular and cerebellar phenomena were absent. There were no sensory or reflex changes; there was some hypertension of the fluid, with a little increase in albumin; the Queckenstedt sign was normal, and the ocular fundi were normal. As bulbar myasthenia gravis or a tumor near the foramen magnum was suspected, the patient was treated by roentgen therapy and died three months later of deglutition bronchopneumonia. Necropsy revealed a fibrillary astrocytoma, the size of a nut, rising from the lower part of the floor of the fourth ventricle.

Chronic Posttraumatic Subdural Hematoma: Roentgenographic Aspect. J. Dereux and A. Hayem.

Various methods have been used to determine the location of a subdural hematoma, including cerebral puncture, ventricular puncture, ventriculography and, best of all, encephalography. Nevertheless, a chronic subdural hematoma may give definite roentgenographic evidence of its location without such procedures. A middle-aged farmer who received a kick by a horse in the frontal region but did not lose consciousness suffered violent headache two months later, together with other symptoms of intracranial hypertension and sluggishness of

thought. On roentgen examination an area of increased density was plainly evident in the left frontal region. Craniectomy in this region revealed a large clot, which was evacuated without difficulty, and the patient recovered in three weeks. Such visualization of the clot must be unusual but is naturally of great value, since encephalography in such a condition is fraught with danger.

LEFT FRONTAL MENINGIOMA WITH GENERALIZED EPILEPTIC SEIZURES FOR FOURTEEN YEARS AND ALMOST COMPLETE ABSENCE OF FOCAL SIGNS AND INTRACRANIAL HYPERTENSION; OPERATIVE CURE. TH. DE MARTEL and J. GUILLAUME.

The mental symptoms presented by a man, aged 33, were rather marked, particularly depression. Scarcely any neurologic signs were present. The roentgenographic aspect of the skull, however, was suggestive, and the ventricular deformation was positive. The tumor, when removed, weighed 170 Gm. The patient was able to walk in eight days. From the practical point of view, the value of an encephalogram even in such long-standing cases is evident.

TUMOR OF THE THIRD VENTRICLE; TRANSCALLOSAL APPROACH AND PARTIAL REMOVAL. TH. DE MARTEL and J. GUILLAUME.

A man, aged 27, complained of sudden diffuse headaches with paroxysmal vomiting and, a month later, with diplopia, which did not yield to treatment for sinusitis. Tremor of the hands prevented finer movements. A new violent headache with contraction of the muscles of the neck provoked further study. Lumbar puncture caused the diplopia to disappear but was followed by projectile vomiting and violent headache, after which choked disk was noted. There was bilateral mydriasis with almost complete absence of pupillary reaction and weakness in associated upward movements and in convergence. The region of the quadrigeminal plate was thus incriminated. Ventriculography revealed symmetrical ventricular dilatation, the third ventricle being dilated in its anterior part but having a filling defect in its posterior portion, indicating a tumor, probably springing from the pineal region.

Operation under local anesthesia in the sitting position was begun by draining the ventricles, and the tumor was approached through the corpus callosum in its posterior portion. The tumor was removed by the cautery loop without loss of consciousness by the patient. After operation, the systolic pressure rose to 220 mm. and the temperature to 103 F., but ventricular tapping quickly brought these values back to normal. Within a few days the ocular symptoms had practically disappeared. The tumor proved to be a fibrillary astrocytoma with a small xanthochromic cyst. There was no obvious disturbance referable to the callosal section.

GLOSSOPHARYNGEAL NEURALGIA; JUXTABULBAR NEUROTOMY (DANDY'S METHOD). A. BAUDOUIN, D. PETIT-DUTAILLIS, R. CAUSSÉ and M. DEPARIS.

A woman, aged 60, complained of attacks of pain in the left ear and pharynx, occurring on drinking or eating and stopping spontaneously after several seconds. There was an intermission for two years with recurrence from time to time after that, but the intensity had become aggravated till even speaking or swallowing saliva produced agonizing pains. The salivary secretion was sometimes abundant, sometimes diminished, and objective examination revealed nothing. Cocainization of the left tonsil and base of the tongue abolished the attacks temporarily, but injections of procaine hydrochloride had no effect. Attacks could always be provoked by touching the tonsillar region. Following suboccipital craniectomy the glossopharyngeal nerve was isolated from the pneumogastric and sectioned without difficulty, with complete relief. Following operation the patient noted a bitter taste on swallowing. A month after operation, sensory

tests within the mouth showed a little diminution to touch on the left side of the tongue. Hot and cold were felt on the left, but taste was lost posteriorly. The palate deviated slightly toward the right, and the left pillar was paralyzed. The posterior wall of the pharynx contracted normally. The sensory disturbances diminished with the passage of time.

FAMILIAL DIFFUSE PERIAXIAL ENCEPHALITIS (SCHILDER'S DISEASE): TWO CLINICAL OBSERVATIONS. G. HEUYER, CLAIRE VOGT and ROUDINESCO.

Few observations of familial diffuse sclerosis have been published in France, although this condition is well known in other countries. The two patients presented came from the same family, one aged 4 years and the other aged 2½. Both had been normal at birth and up to nearly 2 years of age. The condition began with convergent strabismus and nystagmus, followed by gradually increasing spasticity and tremor of the hands and finally by contractures, intellectual deficit and loss of speech. Tonic spasms with continual crying and bilateral optic atrophy completed the clinical picture. The serologic reactions were normal, but from 5 to 7 lymphocytes per cubic millimeter were present in the spinal fluid. The condition resembles most that described by Krabbe and by Symonds. Other children in the family, aged 10 years, 6 years and 4 months, respectively, were healthy. The ancestry seemed normal. The pathogenesis is obscure.

MENINGIOMA OF THE SMALL WING OF THE SPHENOID. PIERRE PUECH and GUY LOISEL.

A case of meningioma of the small wing of the sphenoid is reported on account of the great size of the tumor and the marked hyperostosis produced by the growth in this region. A man, aged 30, had had paralysis of the face of central type for a year and a half. Several months later, headaches began; they lasted an hour or two in the morning. A year after the onset, there developed disturbance in memory, change in character, insomnia, headache and vertigo. Vision in the left eye became reduced; there were diplopia and attacks of complete blindness. Shortly before entering the clinic some aphasic manifestations were found, followed by a generalized convulsion with marked increase in symptoms and choked disk. In eating or drinking the patient had to place himself on all fours and bend his head forward. There were instability, with titubation, adiadokokinesis on the left and plantar extension on both sides. Vestibular tests gave normal results. The spinal fluid showed increased albumin, with 12 lymphocytes per cubic millimeter; ventriculography showed the left foramen of Monro closed and the third ventricle thrust to the right, and roentgenograms of the skull showed marked bony overgrowth of the left pterion. The pulse rate was consistently below 50.

Under local anesthesia, the tumor was exposed and removed in pieces by electrocoagulation, the osteoma being snipped off. The procedure required eight hours, and three transfusions were given; at the end the patient was conscious and neither hemiplegic nor aphasic. The large quantity of wax necessary to stop the bleeding in the bone caused an infection of the wound, but the neurologic symptoms gradually subsided except for marked loss of vision on the left side. Histologically the tumor was a lamelliform meningo-exothelioma (Hortega).

HYPERTHYROID COMA. J. C. MUSSIO-FOURNIER and A. LOUBEJAC, Montevideo.

Nervous disorders accompanying diseases of the thyroid gland are fairly common, but coma of hyperthyroid origin is usually found only in the serious types and during the acute crises of thyrotoxicosis. Coma may occur spontaneously or following an infection or operation or even radiotherapy. Most of such patients succumb. From the metabolic standpoint, albumin and casts are present, but without nitrogen retention or arterial hypertension; the amounts of calcium, potassium, magnesium and dextrose in the blood show no change, but the iodine content is diminished.

A woman, aged 33, had two such crises, but recovered. Following preliminary radiotherapy there were marked tachycardia, fever and oliguria, with rapid emaciation, vomiting, diarrhea and finally convulsions and coma. Intravenous injections of ouabain and lobeline diminished the convulsions, and improvement followed hypodermic injection of dextrose and insulin and enemas containing 30 drops of compound solution of iodine. The condition was improved previous to operation, but on the day following the operation the patient again went into coma and was revived by the same treatment.

Zondek lost nine of thirteen patients with this condition. Disturbance in the liver seemed to be of considerable significance in the case reported here, and the injection of dextrose and insulin seems to be the most rational therapy.

Cerebral Pseudotumor Complicating Hemolytic Icterus. J. C. Mussio-Fournier and A. Carra.

In certain diseases of the hematopoietic system, intracranial hypertension may occur, suggesting cerebral tumor, but the nervous disorders disappear parallel with the amelioration of the blood picture. In a woman, aged 30, hemolytic icterus appeared in 1926, with much urobilin in the urine. The patient suffered from headaches and amblyopia with marked bilateral papilledema, which cleared up following spontaneous improvement of the icterus. Another crisis occurred in 1928, accompanied by projectile vomiting, vertigo and staggering. Aside from high pressure of the spinal fluid nothing significant was observed, and treatment by liver extract brought about improvement both in the blood and in the nervous system. There was probably a serous meningitis occasioned by the blood dyscrasia similar to what one may find in chlorosis and in polycythemia.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 15, 1933

A. C. Buckley, M.D., *President, in the Chair*

Jacksonian Epilepsy and Pathologic Changes in the Skull. Dr. Alfred Gordon.

Localized cerebral irritation leading to contralateral convulsive seizures may be manifested in several forms, such as: (1) attacks with loss of consciousness, (2) attacks without loss of consciousness and (3) attacks with myoclonic or choreiform movements, which may precede, follow or occur in the intervals between the convulsive seizures. Koshevnikoff, in 1894, isolated a variety of the third form known as "epilepsia partialis continua" in which he observed prolonged myoclonic movements, confined to one extremity, on the side of the convulsions. The case described here presents an example of this form of focal epilepsy, but with only occasional myoclonic phenomena in one of the extremities affected with epileptic convulsions. Variations in the duration of myoclonic movements in Koshevnikoff's disease have been observed by other writers.

H. N., aged 44, a driver, stated that seven years ago, for the first time, he had a series of convulsive seizures confined to the left side of the body, involving the face, arm and leg, without loss of conciousness. For several years, he claimed, he was well. About six weeks ago the attacks returned, at which time he first came under my observation. During the course of a half hour he had at least eight or ten attacks. They consisted of rapid clonic contractions of the left leg and arm and slight contractions of the muscles of the left side of the face. During the attacks the affected limbs were in flexion and very spastic. The duration of the

attacks was from twenty to thirty seconds. Consciousness was preserved throughout, but the patient was very pale and complained of headache and dizziness. During the attacks an extensor plantar reflex was in evidence, as well as ankle clonus. Attempts at examination brought on the attacks. Although the patient's mentality was good, he was somnolent and slow in answering questions. For six or seven days he complained of severe headache, but this gradually subsided. The dozen seizures which he had at that time produced weakness in the left arm and leg, which finally disappeared.

Examination gave essentially negative results. A roentgenogram of the skull showed a deeper sella turcica than normal. It measured 7 by 13 mm. There were two areas in the right frontal region showing absorption of bone.

During six or eight weeks of observation, the patient has had no convulsive seizures, but on several occasions he has experienced myoclonic movements in the left hand of one minute's duration.

The roentgenographic picture of the skull suggested four possibilities: (1) trauma of the skull, (2) syphilis, (3) myeloma and (4) a metastatic malignant growth. The first possibility was eliminated, as there was no history of injury of any kind. As to metastasis, a rigorous survey of all viscera and tracts, including the prostate, gave negative results. Syphilis and myeloma were the only two possibilities. Roentgen examination of the long bones showed marked thickening and periosteal proliferation in the upper half of the right tibia, giving the latter a saber-like appearance. A similar process was seen in the right ulna. There was also some irregularity in the upper portion of the left ulna. Such a condition is characteristic of syphilitic osteitis and periostitis. The absorption of the cranial bone is also typical of syphilis.

The localization of the bony process in the right frontal region suggests an irritation of the motor area of the cortex resulting in convulsive seizures of the left arm and leg. The seizures are characterized by rapid clonic spasms in rigid limbs, without loss of consciousness, and myoclonic muscular contractions on the same side in the intervals between the convulsive attacks. These suggest strongly the possibility of Koshevnikoff's "epilepsia partialis continua"; in his original description, the myoclonic twitchings were continuous. Nevertheless, it is rare to find such a condition in its pure form. The cases described subsequently by various observers presented some deviation from Koshevnikoff's original description.

As to the pathogenesis and pathology of Koshevnikoff's form of epilepsy, opinions are divided. Koshevnikoff was in favor of a cortical localization. In Osokin's case, the basis of the disorder proved to be meningo-encephalitis. In Krumholz's case the symptoms were due to encephalitis, with a sclerotic process in the motor cortical region (*J. Nerv. & Ment. Dis.* **40**:17, 1913). Omorokow found in his 30 cases in which operation was performed a marked hyperemia of the corresponding cortex. Wilson and Winkelman also found inflammatory changes in the cortex. In addition to these cortical changes mentioned by some authors, there are observations which indicate involvement of the extrapyramidal segments. In the case described here the lesion of the cranium, strictly localized to the frontal region, indicates cortical irritation of the motor area, but in view of the myoclonic phenomena which appear occasionally between the epileptic seizures, the extrapyramidal system may also be in a state of irritation. To say which is the primary seat and which the secondary is not possible.

DISCUSSION

DR. WILLIAM G. SPILLER: When, in 1908, I observed epilepsia partialis continua in a patient in my service in the Philadelphia General Hospital, I recognized that he had a form of epilepsy which was not generally known. After studying the literature I discovered that the disease had been observed by Russian physicians and I was obliged to depend on German abstracts of the papers by these men (Koshevnikoff, Muratoff, Bechterew and Orlowski). Oppenheim mentioned the condition in a few lines in his textbook. In German abstracts I learned that

Choroschko, in 1907, had reported 3 cases of his own and referred to 11 in the Russian literature. Outside of Russia, Bruns alone had observed this rare type. No information as regards the pathologic condition had been obtained either at operation or at necropsy. Choroschko disagreed with Koshevnikoff in that he believed the lesion to be subcortical. All the information obtainable and the report of my case were published in *The Journal of the American Medical Association* (**52**:1921 [June 12] 1909).

The patient was a young Negro who had syphilis, convulsions confined to the right side of the body and paralysis of the right side of the face and of the right upper limb. Independently of the typical attacks, he had almost constant twitching movements of the right side of the face. The movements lasted several weeks; then similar movements began in the right upper limb, with some displacement of this limb. Dr. Edward Martin, at my request, operated over the nerve centers for the face and upper limb in the motor cortex and removed a portion of gummatous tissue from this part.

This was evidently the first case in which any pathologic condition had been determined and the cortical origin of the continued twitchings established. The French and English literature contained no reference to epilepsia partialis continua so far as I could determine.

A Case of Aneurysm of the Cervical Cord: Pathologic Specimen. Dr. Robert A. Mathews and Dr. George Wilson.

This article will be published elsewhere.

Significance of a Positive Colloidal Gold Curve in Nonsyphilitic Diseases: A Clinicopathologic Study. Dr. Helena E. Riggs.

The work reported here was originally undertaken with Dr. C. S. Potts, who used the clinical diagnosis as the basis of the interpretation of the gold curves. Shortly after his death, several cases included in the series came to autopsy. In these, the clinical diagnosis was so at variance with the results of the postmortem examination that the work was revised. Following the publication of the paper of Potts and Drake on the significance of the gold curve in multiple sclerosis, my attention was drawn to 2 patients with negative serologic reactions and positive gold curves. The clinical signs precluded the diagnosis of multiple sclerosis, while the only indication of a syphilitic etiology was the positive gold curve.

The first patient, a white woman aged 60, was admitted to the Philadelphia General Hospital to the service of Dr. Michael Burns. She gave a history of progressive weakness of the lower extremities for the preceding four years. On admission there was spastic paralysis of the lower limbs, with increased reflexes and a bilateral Babinski sign. There were loss of sphincter control and abolition of all forms of sensation except touch in the legs. Serologic tests of the blood and spinal fluid were negative, but there was a colloidal gold curve of 122333322. After eight months of antisyphilitic treatment there was no clinical improvement. After the period of treatment, the curve read 3455554322. Four and a half years later, the patient died, and an extramedullary endothelioma of the twelfth dorsal segment of the spinal cord was found at autopsy.

The second patient, a Negress, aged 47, was admitted to the service of Dr. J. B. Carnett at the Philadelphia General Hospital. Her complaint was of weakness, vomiting and pain in the back. Examination showed stiffness of the neck with a positive Kernig sign; in the hospital there developed flaccid paralysis of the legs and loss of sphincter control. Progressively, there developed bilateral ophthalmoplegia, bilateral peripheral facial palsy and bilateral involvement of the motor branch of the fifth nerve. Serologic tests were negative with the blood and spinal fluid, but there was a positive curve of the paralytic type. The patient was given vigorous antisyphilitic treatment, but grew worse. Ten months after admission she died. Autopsy showed widespread metastasis from a primary carcinoma of the breast. There was extensive meningeal involvement.

The cases of these patients forcibly demonstrate that a positive gold curve in the presence of negative serologic reactions does not necessarily indicate syphilis of the central nervous system.

A review of the literature revealed that between 37.5 (Moore) and 77.7 per cent (Chevassut) of cases of multiple sclerosis presented positive gold curves.

The incidence of positive curves in conditions other than syphilis or multiple sclerosis was first reported by Lowery in 1912. These were found in routine studies in psychiatric cases. The author reported the finding but placed no great significance on it.

From that time, numerous authors have reported positive curves in cases of meningitis, tumors of the brain and spinal cord and many organic nervous conditions. No attempt was made to explain the phenomenon until Cockrill, in 1925, after studying 151 cases of all types of disease, concluded that "a colloidal reduction may occur in any pathologic spinal fluid."

This study was attempted to determine whether, as Lowery suggested, a positive curve was only an accidental occurrence in nonsyphilitic diseases, or whether, as Cockrill stated, a positive curve might occur with any pathologic spinal fluid. The colloidal gold curves obtained at the Philadelphia General Hospital in 1926 and 1927 were examined. Of 5,289 tests performed, there were 236 positive curves associated with negative serologic reactions in the blood and spinal fluid but no history or clinical evidence of syphilis.

Owing to the fallibility of clinical diagnosis, the investigation was limited to 70 cases in which gross and microscopic examination was made at autopsy. The wisdom of this decision is evident from a study of the tables. A precipitation of more than the first degree in any one tube was considered to constitute a positive curve. An attempt was made to control the personal factor by having one person prepare and standardize the gold solution and read the tests. Following the terminology of Warwick and Nixon, three types of curves were recognized: (1) paralytic (5555543210), (2) syphilitic (0002331000) and (3) meningitic or irritation (00001331).

The cases fell into four groups: (1) Infections directly involving the central nervous system (table 1). This group consisted of 17 cases including acute meningitis, tuberculous meningitis and acute poliomyelitis. There were 4 paralytic curves, 12 syphilitic and 1 meningitic. (2) Systemic toxemias and infections (table 2). In this group are listed 13 cases, including infections of the blood stream, leukemia and chronic nephritis with uremia. There were 3 curves of the paralytic type and 12 of the syphilitic type. (3) Cerebral vascular lesions (table 3). This group comprised 22 cases, including epidural hemorrhage, intracerebral hemorrhage, acute cerebral thrombosis, multiple old thrombotic lesions and cerebral arteriosclerosis. There were 8 curves of the paralytic type and 14 of the syphilitic type. (4) Neoplasms of the central nervous system and vertebrae (table 4). This group, consisting of 18 cases, included primary and metastatic tumors of the brain, spinal cord, meninges and vertebrae. There were 6 paralytic curves, 11 syphilitic and 1 meningitic.

These four groups cover many disease processes, but all have a common factor: The nature of the pathologic physiology is such that products of abnormal metabolism of nerve tissue enter the spinal fluid. It is the presence of such products I believe that influences the precipitation of the colloidal gold.

In group 1 it is possible that the presence of bacteria or the products of bacterial metabolism may be responsible for the precipitation. If this is true, there is no relation between the degree of precipitation and the quantitative alteration of the spinal fluid, since in tuberculous meningitis, in which bacteria and bacterial products are few, curves of greater magnitude were found than in purulent meningitis. I believe that a more logical explanation is that the bacterial products act as an irritant to nerve tissue, resulting in abnormal metabolic products of the nerve tissue entering the spinal fluid through the blood and lymph drainage into the subarachnoid space.

TABLE 1.—*Colloidal Gold Curves Obtained in Group 1*

Case	Colloidal Gold Curve	Pathologic Diagnosis	Clinical Diagnosis
1	2222333100	Sinus thrombosis, acute meningitis	Acute meningitis
2	1112210000	Acute meningitis	Tuberculous meningitis
3	4443321100	Acute meningitis	Acute meningitis
4	0012100000	Acute meningitis	Acute meningitis
5	1111122211	Acute meningitis	Arteriosclerosis
6	0012100000	Tuberculous meningitis	Syphilitic meningitis
7	1111222200	Tuberculous meningitis	Tuberculous meningitis
8	4444444444	Tuberculous meningitis	Tuberculous meningitis
9	2222210000	Tuberculous meningitis	Acute encephalitis
10	1111221100	Tuberculous meningitis	Cerebral thrombosis
11	1223332200	Tuberculous meningitis	Tuberculous meningitis
12	0000013333	Tuberculous meningitis	Tuberculous meningitis
13	0012210000	Tuberculous meningitis	Typhoid meningitis
14	2222332000	Tuberculous meningitis	Tuberculous meningitis
15	0001221000	Tuberculous meningitis	Embolic hemiplegia
16	3333421000	Tuberculous meningitis	Uremia
17	1122210000	Poliomyelitis	Poliomyelitis

TABLE 2.—*Colloidal Gold Curves Obtained in Group 2*

Case	Colloidal Gold Curve	Pathologic Diagnosis	Clinical Diagnosis
1	1112110000	Subacute bacterial endocarditis	Syphilitic hemiplegia
2	2223331000	Subacute bacterial endocarditis	Meningeal irritation
3	0011222100	Subacute bacterial endocarditis	Tuberculous meningitis
4	1111122221	Hodgkin's disease	Uremia
5	1122200000	Myelogenous leukemia	Myelogenous leukemia
6	4444331100	Chronic glomerular nephritis	Uremia
7	4444321000	Chronic glomerular nephritis	Uremia
8	1233210000	Chronic glomerular nephritis	Uremia
9	0012210000	Chronic glomerular nephritis	Uremia
10	2223221000	Chronic glomerular nephritis	Uremia
11	1122210000	Chronic glomerular nephritis	Uremia
12	5554321000	Chronic glomerular nephritis	Uremia
13	0012210000	Chronic glomerular nephritis	Uremia

TABLE 3.—*Colloidal Gold Curves Obtained in Group 3*

Case	Colloidal Gold Curve	Pathologic Diagnosis	Clinical Diagnosis
1	0011221100	Intraventricular hemorrhage	Cerebral hemorrhage
2	1111120000	Cerebral hemorrhage	Cerebral hemorrhage
3	2333221100	Cerebral hemorrhage	Cerebral tumor
4	0233200000	Subarachnoid hemorrhage	Cerebral thrombosis
5	2233221000	Subarachnoid hemorrhage	Cerebral hemorrhage
6	0012220000	Cerebral hemorrhage	Cerebral hemorrhage
7	1122200000	Meningeal hemorrhage	Cerebral hemorrhage
8	0011221000	Meningeal hemorrhage	Uremia
9	1112100000	Cerebral thrombosis (acute)	Myocarditis
10	1112100000	Cerebral thrombosis	Cerebral thrombosis
11	4443332221	Cerebral thrombosis	Cerebral neoplasm
12	1122100000	Cerebral thrombosis	Cerebral thrombosis
13	2244444431	Cerebral thrombosis	Uremia
14	2222223221	Cerebral thrombosis	Cerebral thrombosis
15	1123311000 3444321000	Multiple cerebral thromboses	Cerebral edema
16	3333330000	Multiple cerebral thromboses	Cerebral thrombosis
17	1112220000	Multiple cerebral thromboses	Pseudobulbar palsy
18	1223410000	Multiple cerebral thromboses	Cerebral thrombosis
19	4444331100	Multiple cerebral thromboses	Epidural hemorrhage
20	1122110000	Multiple cerebral thromboses	Cerebral thrombosis
21	3333334432	Multiple cerebral thromboses	Senile dementia
22	2333333300	Multiple cerebral thromboses	Uremia

In group 2, the explanation is similar to the foregoing one. The toxins of the systemic toxemia or infection serve as an irritant to the nerve tissue and the resulting abnormal products alter the make-up of the spinal fluid. Or, if the statement of Matz and Novik is accepted, that systemic toxemias and infections cause a change in the spinal fluid, this change may be a factor in the precipitation of the colloidal gold.

Group 3 gives the strongest proof that the gold curve results from alteration of the spinal fluid by abnormal metabolic products of nerve tissue. In this group, products of cell destruction from hemorrhage, thrombosis or ischemic cell disease are emptied into the spinal fluid by the blood and lymph channels draining the subarachnoid space. I consider that multiple sclerosis belongs in this class. The progressive destruction of the myelin sheaths would result in degenerated nerve tissue being carried into the spinal fluid.

Group 4, that of neoplasms, suggests several interpretations for the precipitation of the colloidal gold. Neoplasms expand at the expense of the tissue around them

TABLE 4.—*Colloidal Gold Curves Obtained in Group 4*

Case	Colloidal Gold Curve	Pathologic Diagnosis	Clinical Diagnosis
1	1223333333 3444433333	Endothelioma of the spinal cord	Amyotrophic lateral sclerosis
2	3455543322	Endothelioma of the spinal cord	Syphilitic myelitis
3	2221100000	Metastatic tumor of the meninges	Tuberculous meningitis
4	3443321000	Metastatic tumor of the meninges	Metastatic tumor of the brain
5	3344333222	Metastatic tumor of the meninges	Syphilis of the central nervous system, tuberculous meningitis
6	0000233333	Metastatic tumor of the vertebrae	Metastatic tumor of the vertebrae
7	0000112100	Metastatic tumor of the vertebrae	Metastatic tumor of the vertebrae
8	2333221000	Glioma of the cerebellum	Encephalitis
9	1112100000 2234321000	Glioma of the cerebrum	Tumor of the brain
10	1111122100	Meningioma of the frontal lobe	Tumor of the brain
11	0112333210	Glioma of the temporal lobe	Cerebral thrombosis
12	2222331000	Glioma of the basal ganglia	Cerebral thrombosis
13	1111222000	Glioma of the occipital lobe	Tumor of the brain
14	0000122100	Fibroma of the acoustic nerve	Multiple sclerosis
15	4454432110	Meningioma of the frontal lobe	Epilepsy, syphilis of the central nervous system
16	2233344222	Metastatic tumor	Tuberculous meningitis
17	1232110000	Metastatic hypernephroma	Metastatic hypernephroma
18	2234321000 1112100000	Sarcoma of the scalp, meningeal metastasis	Cerebral gumma

and so permit the degenerated nerve tissue to enter the spinal fluid. The tumor cells, per se, may act as an irritant to the cerebral tissue, either in the rôle of a foreign body or through the abnormal metabolism of the tumor cell.

As a result of this analysis, the conclusion is reached that the precipitation of colloidal gold is dependent on changes in the spinal fluid resulting from the presence of abnormal metabolic products of the nerve tissue. A type of curve specific for any disease or even for any one of the four groups was not found. In the series there were 21 paralytic curves, or 30 per cent; 47 syphilitic curves, or 67.1 per cent, and 2 irritation or meningitic curves, or 2.8 per cent. Paralytic curves were present in cases of acute poliomyelitis, acute and tuberculous meningitis, neoplasms of the central nervous system, cerebral vascular disease and uremia. In 16 cases of acute and tuberculous meningitis, the so-called meningitic curve appeared but once, and that in a case of tuberculous meningitis. In 11 cases of cerebral neoplasm the paralytic curve was present twice and the syphilitic 9 times.

These observations are not in accord with Chevassut's statement that the gold curve is specific for a given disease and that the form of curve is related to the causative factor of the disease. Her observations were based on the curves in 189 clinical cases of multiple sclerosis, in which 70 per cent gave a specific curve of the type 000322200. In refutation the curves are presented in 24 cases of multiple

sclerosis in which the diagnosis was proved by autopsy and confirmed by microscopic study (table 5).

This series was studied at the Philadelphia General Hospital between 1921 and 1932. There was a positive curve in 62.5 per cent, or 15 cases. Of the positive curves, 53.3 per cent were paralytic and 46.7 per cent were syphilitic. None of the curves were identical, and there was a variation in repeated curves in the same case. I have introduced this material at this point to show that in a single condition such as multiple sclerosis there is no evidence of a specific curve. Further, in 67.1 per cent of widely different cases I obtained the type of curve which has been claimed as specific for multiple sclerosis.

Other investigators support the contention that the type of colloidal gold curve cannot be considered as a criterion of differential diagnosis. Vogel found syphilitic curves in cases of acute poliomyelitis, tuberculous meningitis, transverse myelitis, chronic nephritis, chronic alcoholism, tumor of the brain and arteriosclerotic hemiplegia. He noted that of cases of acute poliomyelitis, 1 showed a paralytic curve and 2 a syphilitic. Davis and Krause, investigating acute epidemic

TABLE 5.—*Colloidal Gold Curves in Cases of Multiple Sclerosis Confirmed by Autopsy (1921-1932)*

Negative Curves 9 Cases	Paralytic Curves 8 Cases	Syphilitic Curves 7 Cases
	(1) 5555542100	(1) 2234442000
	(2) 4221000000	(2) 0111100000 2233442100
	(3) 4444311000	(3) 1112222000
	(4) 4444332100	(4) 1112110000
	(5) 2222221000 3332110000	(5) 0012110000
	(6) 5554431000 4544443311 4443211000	(6) 1223340000
	(7) 5554433000	(7) 2234321000
	(8) 3444421100	

encephalitis, concluded: "There is no relationship between the presence of a positive curve and the severity of the disease, its duration, or the clinical picture."

To summarize, from the analysis of the colloidal gold curves in 70 nonsyphilitic cases, the following conclusions are reached: 1. The precipitation of colloidal gold is related to the presence of products of abnormal metabolism of nerve tissue in the spinal fluid. 2. There is no relationship between the type of curve and the pathologic process involved.

DISCUSSION

DR. B. J. ALPERS: Was any attempt made to analyze the ratio of globulin to protein in the spinal fluid in order to determine whether this may be a factor in the production of abnormal gold curves?

DR. HELENA E. RIGGS: I deliberately did not take up the subject of the chemistry of spinal fluid showing a positive gold curve. I have reviewed the work done by chemists on spinal fluids showing positive gold curves, but the consensus at present is that the chemical change in the fluid responsible for precipitation of colloidal gold is unknown. The spinal fluid has been studied from the point of view of increased globulin, change of the ratio of albumin to globulin and the unit of euglobulin present and from the point of view of change in the spectroscope. No one chemical change in the spinal fluid covers all the cases in which there is a precipitation of colloidal gold. Chemists believe that an analysis of brain tissue in cases of positive gold curves and of normal brain tissue may throw some light on the subject.

CONCEPTS IN THE TREATMENT OF CHOREA. DR. JOSEPH B. WOLFFE and DR. PAUL SLOANE.

It is fairly well recognized that Sydenham's chorea is not an independent disease, as was thought for many years, but a part of rheumatic disease. It is most important to appreciate this fact fully for the following reasons: 1. Recognition of the premonitory symptoms of chorea, both somatic dysfunction and nervous instability, in children will eliminate the possibility of these children being dismissed as neurotic and spoiled. 2. Following the acute stage of chorea, involuntary movements may persist for many months. This may be residual chorea due to cellular pathologic changes as a result of rheumatic disease which has been arrested. On the other hand, it may be chronic chorea due to a smoldering infection which is diminished but not destroyed. There are certain criteria by which chronic chorea can be distinguished from residual chorea. Thus, patients with chronic chorea, because of a latent persistent disease, usually show apparent anemia. They are apt to suffer from moistness of the hands, epigastric pain, headaches, recurrent sore throat, marked fluctuation in daily temperature and pains in the joints or muscles. They have a fixation of weight, so that they neither gain nor lose. Slight leukocytosis is the rule. They tend to show progressive abnormal signs, such as gradual cardiac enlargement (judged from the teleroentgenogram and not from physical examination), adventitious sounds such as murmurs, pericardial frictions and occasional electrocardiographic changes such as P R delay, ectopic beats, slurring of the initial ventricular complex and changes in the T wave. Mentally, these children are exalted, cheerful and very active. Enuresis is common. Patients with residual chorea, on the other hand, show none of these signs. In these cases, if a cardiac lesion such as mitral insufficiency, plastic pericarditis or myocardial change occurs during the period of chorea, the size of the heart remains fixed. The abnormal physical signs persist for a long time and do not vary from one examination to another.

What is most important of all is to recognize carditis as part of the picture of rheumatic disease, rather than a sequel or complication. By recognizing chorea as only a manifestation of rheumatic disease one thinks less of the involuntary purposeless movements and more of the possible cardiac damage. The treatment, therefore, is essentially that for active rheumatic disease, in which the purposeless movements are only one of the troublesome manifestations. While many consider chorea terminated when the purposeless movements disappear and therefore look on it as self-limited, this is really not the case from a cardiac point of view. When the major manifestations of chorea are over and the child is carefully watched, one frequently finds progressive cardiac damage, which makes its appearance gradually and leads to definite cardiac crippling. This in itself, as has already been pointed out, shows that the active rheumatic process has been going on despite the fact that the symptoms of chorea have greatly subsided. We believe that the reason for many cases of irreparable damage of the heart lies in the fact that many physicians feel at ease as soon as the choreic manifestations clear up, without following up the case for many months. At Temple University Hospital and Willow Crest Convalescent Home a patient with chorea is not discharged until two years following the active stage of the disease.

With this fundamental conception of chorea, the aim of treatment is to shorten the course of the rheumatic disease and minimize its evil effects. During the acute stage absolute rest is essential. A thorough neurologic and physical examination, with particular attention to the cardiovascular system, is important. Electrocardiographic and teleroentgenographic studies should be done if possible. This completes the initial record and serves as a standard for further comparative studies in a given case. In this way minor cardiovascular changes may be recognized months and sometimes years before physical examination alone might reveal them. Sedation in the form of warm baths, phenobarbital and bromides, administration of iron compounds and cod liver oil, removal of foci of infection and the administration of salicylates are first tried. When these means fail to control the choreiform movements, other remedies are utilized, such as neoarsphenamine, sodium cacodylate, foreign proteins and typhoid vaccine. Every one of these methods has its champion.

Phenyl-ethyl-hydantoin, which we have employed for the last two years, has proved of value in some stubborn cases. In our experience, we may conservatively estimate that this drug was effective in about 50 per cent of cases, while in the remainder there was no effect.

After the acute chorea has subsided, or in cases in which there has been no acute stage, the patient is kept in an institution or at home for many months. He is watched carefully for any other manifestations of rheumatic activity, such as recurrent sore throat, pains in the joints or muscles, and progressive abnormal cardiac signs as judged by physical examination, teleroentgenograms and electrocardiograms. Immediately on the appearance of such manifestations of rheumatic activity, the child is kept at rest for a still longer period. All of our patients are immunized with an autogenous vaccine prepared from the secretions of the nose and throat. In this way we aim to prevent recurrences and hope that rheumatic heart disease will be largely prevented.

DISCUSSION

DR. J. C. YASKIN: Dr. Sloane and Dr. Wolffe have raised a number of old and interesting problems. It has long been recognized and frequently emphasized that chorea is part of a general infection in which the heart is frequently implicated. Yet, as one treats a great many patients in hospitals, the feeling is gained that infection is not the only element in the etiology and in the management of chorea. The real problem, indeed, is not the treatment of the individual attacks but the prevention of recurrences. I have repeatedly seen cases in which all foci of infection have been cleared up in which there has been no endocarditis but in which there have been repeated attacks of chorea, often twice a year. One, therefore, gets the impression that the infection is not the only element in the etiology of chorea, and one is compelled, with Oppenheim, to concede that psychogenic elements play a large rôle both in the production and in the recurrence of attacks.

Chronic Sydenham's chorea, if it exists at all, is an extremely rare disease. Most of the cases of so-called chronic chorea are not chorea to start with, being probably psychoneuroses with choreic movements. The residual symptoms referred to in this presentation are common and are often habit formations residual to the chorea.

I wish to ask the essayists whether they have had an opportunity to follow the reported cases long enough and whether recurrences have taken place under the treatment that they have suggested. The average case of chorea ends in recovery in from four to eight weeks without any specific medication. It would appear that one is not justified in subjecting these children to the risk of this strenuous treatment without a definite reason for doing so. In addition to the complications enumerated by Dr. Sloane, I may add that I have observed a case of optic atrophy following this treatment.

DR. PAUL SLOANE: Our patients have been followed for from two and one half to three years. Phenyl-ethyl-hydantoin apparently does not prevent recurrences, and that is why we administer the vaccine. It is our impression that children treated by this method show fewer complications and recurrences. In regard to psychogenic causes as factors in recurrences, we have had cases in which active chorea has come on following psychic trauma, but on the whole we found that psychic factors did not predominate. A statistical study would be useful in helping us to arrive at some conclusion in this respect. There is reason to believe that if there can be actual recurrence of the rheumatic phenomena there cannot be similar involvement of the brain. Most children who have had chorea are irritable and sensitive and react rapidly to environmental influences. They are frequently apt to present behavior problems, even under comparatively good family circumstances. I wonder if examination of these patients would not show residual damage of the nerve cells. As far as the self-limitation of the disease is concerned, we meant to emphasize the fact that our patients were treated by ordinary methods at first. Phenyl-ethyl-hydantoin was used only when other methods failed. I have never heard of blindness as a result of the treatment. Practically all of the cases with complications reported in the literature have ended in recovery.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

KARL M. BOWMAN, M.D., *Secretary*

Regular Meeting, Dec. 21, 1933

E. S. ABBOT, M.D., *Presiding*

CONTAMINATION OF THE CEREBROSPINAL FLUID BY BLOOD. DR. PHILIP SOLOMON, MISS MARY E. DAILEY and DR. FRANK FREMONT-SMITH.

This article was published in full in the June 1934 issue of the ARCHIVES, page 1222.

DISCUSSION

DR. H. H. MERRITT: This presentation is important since one often sees the statement in the literature that a lumbar puncture was done but the fluid was bloody and could not be analyzed. Dr. Solomon has shown that such fluid can be evaluated. The important thing is an accurate determination of the number of red blood cells in the fluid; from this, as Dr. Solomon has shown, the white cell count and protein content of the fluid (before contamination with blood) can be determined.

ANEURYSM OF THE AORTA, WITH COMPRESSION OF THE SPINAL CORD. DR. ROBERT A. GREGORY.

Although aneurysms of the aorta have been frequently reported, the complicating factor of compression of the spinal cord has been discussed by few authors, in spite of the fact that the combined picture was reported as long ago as 1841. Because of its rarity, the following case is presented:

M. C., a leather worker, aged 51, was first seen in the outpatient department of the Massachusetts General Hospital on July 17, 1930; for a year he had had a dull, aching sensation under the sternum and radiating into both axillae, which he thought was precipitated by eating heavy foods. The usual duration of the symptom at any one time was between three and four hours. Physical examination revealed little of note. The Wassermann reaction of the blood was negative; the reaction to the Hinton test was weakly positive. Three months later the patient was free from symptoms.

On March 6, 1933, the pain returned; it was more severe and radiated as far as the right scapula. He also complained of numbness in both legs, more marked in the left, some difficulty in walking and slight urinary incontinence, all of which had appeared after jumping over a puddle of water. Physical examination revealed a spastic, ataxic paraplegia. On lumbar puncture the initial pressure was 210 mm. of water; it dropped to 30 mm. on withdrawal of 10 cc. of cerebrospinal fluid. Examination of the fluid showed xanthochromia, lymphocytes, a total protein content of 186 mg. per hundred cubic centimeters, a colloidal gold curve of 0123333321, and a strongly positive Wassermann reaction. The patient was given intense antisyphilitic treatment, consisting of iodides, bismuth and neoarsphenamine, in spite of the spinal fluid block. After two months he stated that his legs were stronger and that incontinence had disappeared. Treatment was discontinued because of arsenical dermatitis.

Two weeks prior to admission, the cane he used in walking slipped into a crevice and caused a jolt. From that time he noticed that his legs became progressively weaker and very "jumpy." Five days before entry there appeared incontinence of urine and a feeling of tightness about the lower part of the abdomen. The only additional feature of significance in the history was that he had a primary syphilitic lesion at 21 years of age.

Physical examination revealed that the pupils were equal and slightly irregular and reacted poorly but definitely to light; the heart was enlarged to the left,

with normal dimensions at the base; the blood pressure was 185 systolic and 90 diastolic; the aortic and pulmonary second sounds were equal; there were no thrills, but a soft systolic and early diastolic murmur was heard at the apex. Posteriorly, to the left of the fourth and fifth dorsal vertebrae, there was palpable pulsation, over which the heart sounds were well heard. No spinal tenderness was noted. The urinary bladder was distended. Neurologically, the legs were spastic and weak, and although all movements could be performed the patient was unable to walk. A sensory level was discovered at the ensiform process. Position and vibratory sense were absent in the legs. The abdominal reflexes were present, but diminished; the knee jerks were active, the right more so than the left; the ankle jerks were absent; the plantar reflexes were of the type of dorsiflexion.

A combined cisternal-lumbar puncture showed complete spinal fluid block, with a lumbar fluid similar to that obtained by the previous tapping. Iodized poppy-seed oil 40 per cent was injected into the basal cistern, and a roentgenogram was made of the thorax which revealed an aneurysm of the descending aorta, about the size of a grapefruit, eroding half through the bodies of the fourth, fifth and sixth dorsal vertebrae and arresting the iodized oil at the level of the fourth vertebra.

Surgical intervention being deemed inadvisable, the patient was discharged to a convalescent hospital with a diagnosis of neurosyphilis, aneurysm of the aorta and transverse myelitis.

Review of the current literature indicated the rarity of such cases. In 1924, over five thousand cases of aortic aneurysms were reported. Only eleven cases showed clinical evidence of compression of the spinal cord. The diagnoses, save in cases presenting visible expansile tumors, were made by roentgen or postmortem examination, in most instances by the latter method. In one case, in which a diagnosis of tumor of the spinal cord had been made, the aneurysm was discovered during laminectomy.

The vertebral erosion in all instances was in the upper and middle dorsal regions and resulted in ten of eleven cases, from aneurysmal sacs on the descending loop of the aorta. A second dilatation on the arch, having no connection with the spinal picture, was found in one of these cases. In the remaining case, the sac was at the upper part of the ascending loop and, although erosion was marked, there were no clinical signs of involvement of the spinal cord. Autopsy in this instance disclosed a fibrous tissue infiltration of all the structures surrounding the sac. The vertebrae were involved to such an extent as to leave a mere shell of bone through which a finger was pushed with ease into the spinal canal. This seemed to be the pathologic picture corresponding to a clinical period just prior to the onset of compression of the spinal cord. Some authors reported even more marked vertebral caries, with exposure of the cord itself, in which no clinical signs of compression were present. In other cases with such clinical evidence post-mortem examination disclosed spinal cords with no evidence of gross or microscopic changes, which is difficult to explain. In seven of the cases, however, definite pathologic changes of the cord had been caused either by direct compression from the aneurysm or by epidural hemorrhage through rupture of the sac; there were two examples of the latter. In some instances the dura was adherent, but in no case was it ruptured. Microscopic sections were reported by two writers. In one case there was degeneration of the spinal cord typical of compression; the other showed merely an interstitial edema of the white substance at the site of pressure. In both sections there was evidence of old syphilitic involvement of the cord.

The clinical similarities were as striking as the pathologic changes. All cases were in patients between the ages of 45 and 60. Aneurysmal, substernal or scapular type of pain, which was present in eight of the eleven cases, antedated the onset of spinal symptoms by from one to three years. Paraplegia was present in all but one case, in which there was a well defined Brown-Séquard syndrome. The paralysis was spastic in the majority of cases. In two, however, it was flaccid, one being associated with tabes dorsalis and the other with epidural hemorrhage

and sudden shock to the central nervous system. The onset of paraplegia in about half the cases was gradual; the slowly eroding pressure of the aneurysm might account for this. On the other hand, the rapid onset in the remainder might well be explained by any one of three causes: (1) a slight jolt, which might cause crumbling of the thin shell of bone and allow the aneurysm to press directly on the cord; (2) a sudden release of pressure, by the same mechanism, which might cause rupture of the sac and epidural hemorrhage, and (3) acute kyphosis with a pinching off of the canal and its contents. Paralysis or paresis was accompanied in most instances by a sensory level; in some cases this was more marked than in others, and in one there was no sensory loss.

It is of special note that of eight cases in which the clinical condition of the heart was described there was not one in which it was reported as normal. Only three showed widening at the base as determined by percussion; in three the heart was enlarged, the base being of normal dimensions; in four diastolic murmurs were heard in the aortic region, and in three there were expansile tumors. In short some defect was present to cause at least a suggestion of the presence of an aneurysm. The location of the sacs in these cases tends to limit the cardiologic findings of import to the back. For this reason the aneurysm is often missed clinically, as in the case here presented.

Analyses of the cerebrospinal fluid in this series were neither fully nor consistently reported. When epidural hemorrhage was found, the fluid was tinted with blood. The total protein and cell counts varied from normal to slightly abnormal. Hydrodynamics were not recorded. In a number of cases the examination of the spinal fluid did not indicate more than a partial block (chemically estimated), from which it may be concluded that examination does not always confirm a diagnosis of compression of the spinal cord. This was particularly well demonstrated in one case with marked paraplegia and a definite sensory level in which the fluid was reported as normal in all respects. In such instances it might be advisable to follow the intrathecal introduction of iodized oil by fluoroscopy.

Antisyphilitic therapy in cases of aneurysm with compression of the cord has been consistently unsuccessful. In cases in which improvement has been noticed, it has been transient. The addition of rest in bed would be of twofold value, minimizing the chance of strain on markedly weakened bone and affording the patient the best opportunity to regeneration of bony tissue should the progress of the aneurysm be checked by the medication.

DISCUSSION

DR. J. B. AYER: When Dr. Gregory told me how few cases of compression of the spinal cord from aneurysm were on record I was amazed. Having seen one case early in my medical career, I naturally supposed that it was more common, and yet I now realize that I have never seen another until now. One point of warning, assuming that a given lesion is necessarily the cause of the compression: Dr. Mixter will remember a case of known syphilis of the nervous system with transverse lesion of the cord and complete block in the fluid. The patient was treated intensively for syphilis, without improvement. Operation was undertaken with the idea that we were probably dealing with a gumma. Much to our surprise, a meningioma was found and completely removed. From the therapeutic point of view it seems almost futile to think of doing anything beneficial after the vertebrae have been destroyed and the cord has been compressed. Numerous suggestions were made in this case, but none seemed likely to be of benefit.

DR. T. J. PUTNAM: The diagnosis in this case was made in the first place not from the roentgenograms but by fluoroscopy. It was confirmed by the expansile pulsation in the back. Of course, if there had been a tumor invading the spinal canal there would have been no question.

DR. W. J. MIXTER: In this case particular thought was given to an attempt to find some surgical procedure that would give even a remote chance of success.

My associates and I considered attempting to retract the aneurysmal sac from the vertebral column with the interposition of a bone graft between it and the cord, but this seemed like a rather forlorn hope. The other forms of surgical treatment, such as the use of fascial bands and wiring, were also considered.

A Large Cholesteatoma Over the Left Hemisphere. Dr. Gilbert Horrax.

A woman, aged 43, was relatively normal until she completed high school and entered college. She was unable to continue with the college course and, after trying various kinds of school work, had a nervous breakdown in 1908. Ten years later she began to act queerly, was overactive, noisy and elated and showed a flight of ideas. In this year she entered the state institution, from which she was discharged two years later with a diagnosis of a manic-depressive state. She was admitted to this institution several other times and discharged. While there in 1932, roentgenograms of the skull showed a large calcified shadow in the left hemisphere. For this reason she was referred for surgical treatment of the intracranial mass. There were no other neurologic signs or symptoms.

Operation was performed in two stages, the first on Dec. 13, 1932 and the second on December 16. On the latter date a huge calcified tumor was removed from the left hemisphere, or rather from its intracranial site between the dura and the left hemisphere. After a somewhat stormy convalescence, the patient was discharged with some improvement in her mental status; she reentered the state institution for a brief period and showed steady improvement in her mental condition. After a few months she was able to leave the state hospital and has continued to report there from time to time; her condition has steadily improved over the course of a year.

The tumor which was removed was a cholesteatoma enclosed within a completely calcified shell. The weight of the mass was between 400 and 450 Gm.

DISCUSSION

Dr. W. J. Mixter: I think that Dr. Horrax has done a remarkable piece of work. The cholesteatomas are likely to cause marked deformity of the brain with little symptomatology. I recall a patient with a large, laterally situated cholesteatoma. Every one was much surprised at the size of the tumor in view of the paucity of symptoms. Are these tumors not rare, especially in comparison with cholesteatomas situated in the midline?

Dr. J. B. Ayer: Where did the tumor originate? Was a lumbar puncture done? I recall two cases, in one of which Dr. Horrax operated after a delay of several years; the clinical picture was that of a lesion of the posterior fossa. In another case in which an operation was done by Dr. Mixter, the tumor presented in the middle fossa.

Dr. A. W. Stearns: Is it assumed that there is any connection between this tumor and the mental attack? The classification was manic-depressive psychosis. Did the patient have manic-depressive psychosis and incidentally a tumor? It is well known that tumors are discovered at autopsies at state hospitals in all sorts of mental sickness.

Dr. M. R. Kaufman: About five or six years ago I had occasion to look over the autopsy protocols at the Boston Psychopathic Hospital. There were about 65 cases of tumor discovered at autopsy. The age incidence was toward the arteriosclerotic age period; all cases were diagnosed as arteriosclerotic or senile psychoses. Should one x-ray the occiput of each patient as a routine measure? I do not think that the diagnosis was made before autopsy in any of the cases.

Dr. F. Fremont-Smith: Is it possible that this tumor reached its full size early in life and that the brain, in its growth, adapted itself to the mass rather than that the tumor actively distorted the brain? If this were true it might explain how such a large tumor could be present without giving rise to more signs and symptoms.

DR. G. LAVINE: This patient was not subjected to psychoanalysis. The lesions were on the right and not the left side. Lumbar puncture was not done. It was thought best that puncture be performed under Dr. Horrax's supervision. I think that the tumor was present in early childhood, because it was said that at the age of 2 the patient had a large head and had difficulty in walking.

DR. G. HORRAX: I do not know whether the tumor had anything to do with the patient's mental state or not. All that could be done was to give her a chance; it may be that the psychosis is now in a period of quiescence and that she may have a relapse. If lumbar puncture had been carried out I doubt whether it would have helped in the diagnosis. I do not know the characteristics of the cerebrospinal fluid in cases of cholesteatoma. This tumor occurs about once in every two hundred cases of tumor of the brain. It is a congenital lesion which usually starts in the midline and either stays there or pushes out into the frontal or temporal region. It may be either a dermoid tumor or an epidermoid growth, through the surface of which one can see a "pearly tumor." Sometimes it contains hair and cheesy material, or it may contain other elements and is then known as a teratoma. In the case reported here the growth was a simple epidermoid tumor, the shell of which had become calcified for some reason. This is, of course, an extremely slowly developing growth. Why should patients with such a huge tumor show mental symptoms and no other neurologic signs if the mental symptoms are due to the tumor, as in cases of subdural hematoma? In the latter instance the mental symptoms are caused by the hematoma, and when the clot is removed the mental status becomes normal. As I tapped the cavity from which this tumor was obtained for several weeks I gradually withdrew less and less fluid; I supposed that the hemisphere had expanded and had probably filled out the intracranial space.

MYELOTOMY OF THE COMMISSURE: A NEW METHOD OF TREATING PAIN IN THE UPPER EXTREMITIES. DR. TRACY J. PUTNAM.

Treatment for pain in the abdomen and legs by means of chordotomy is now a well established and satisfactory procedure. Pain in the upper cervical region and in the face can usually be relieved by section of the posterior roots without severe disability. Treatment for pain in the arms and shoulders has, however, not been very satisfactory. Adequate section of posterior roots leaves the upper extremity entirely useless, and it is difficult to perform chordotomy high enough to control pain in the arms. It must have occurred to many neurologists that a patient with intractable pain in the arm or shoulder would be better off if syringomyelia should develop, and the suggestion has doubtless been made that an artificial destruction of the pain fibers as they cross in the decussation about the central canal would be a rational procedure. As far as I know it has never been attempted before. I have carried out this procedure in three instances.

The first case was that of a woman with carcinomatous metastases in both axillae, with extreme pain. The cord was exposed from the fourth cervical to the third dorsal segment and a needle was inserted between the posterior columns at intervals of a few millimeters up and down the exposed area and was maneuvered in such a way as to pass through the commissure. It was found possible in this way to proceed a segment or two beyond the limits of the laminectomy. The patient was not upset by the operation and was completely relieved from pain until death two months later. She showed loss of sensation to pin prick approximately over the fifth to the eighth cervical segment, but practically no loss of tactile or position sense. There was a certain amount of atrophy of the small muscles of the hands, but they were as useful as they had been before the operation. There was loss of vibration sense in the legs, but no ataxia. The perception of pin prick returned a week or two after the operation, but not the spontaneous pain.

The second case was that of a man with pain from carcinomatous metastases, apparently from the lung, which affected both sides of the neck as well as the axillae. The pain was so severe that doses of ¼ grain (0.0162 Gm.) of morphine did not wholly relieve it. A similar procedure was carried out on this patient,

except that a special instrument was used, which appeared to give better results than the needle. In this instance the section of the commissure was carried up to the first cervical segment. This patient showed no atrophy of the hands, but he was not completely relieved from the pain in the upper cervical segments. The pain was, however, dull and burning instead of sharp, and relief was obtained by codeine instead of morphine.

The third operation ended in a fatality and doubtless represents an error in judgment, as well as an error in technic. The patient was a cachectic old woman, almost moribund from carcinomatous metastases to the shoulders and neck, who died following an operation in which the commissure was divided all through the cervical region and the first two dorsal segments. It is probably wiser to section the posterior roots in the segments above the fourth dorsal vertebra on account of the possibility of respiratory difficulties.

Sections from the spinal cord in the first case showed moderate damage to the posterior columns and a satisfactory division of the commissure throughout most of the extent of the operation. At a few levels the anterior horns were affected. The damage should be less with the new instrument.

This operation could be modified to affect only the posterior horns of one side by making the incision ventral to the posterior roots. The precise indications for the operation remain to be established, but it should, on the whole, be safer than bilateral cervical chordotomy and should be free from the danger of paralysis of the bladder.

DISCUSSION

DR. G. HORRAX: Dr. Putnam always brings something new and interesting to these meetings. The operation which he has proposed has certain distinct advantages. The principal difficulty in chordotomy is that one may not go deep enough and will have to repeat it later. Also, in chordotomy there is some discrepancy between the level of the section and the relief from pain. Bailey believes that the disturbances after chordotomy are distinct disadvantages and are difficult to treat.

DR. W. J. MIXTER: I have watched the development of this operative procedure with great interest. It seems to me that Dr. Putnam has brought out something of definite value and originality. It remains for him to improve the instrument which he uses to cut the commissure so that the section can be made without trauma to the rest of the cord. The close proximity of the respiratory centers in the cord adds greatly to the dangers of the operation.

DR. H. H. MERRITT: Dr. Putnam has shown how anatomic knowledge of the pathways of pain can be put to a practical use; by cutting these pain fibers in the commissure he avoids damaging the respiratory tracts. I wish to ask if Dr. Putnam has ever considered cutting the pain fibers as they enter the substantia gelatinosa?

DR. T. J. PUTNAM: In reply to Dr. Merritt, I think that it might be possible in the case of unilateral pain.

KARL BOWMAN, M.D., *Secretary*

Regular Meeting, Jan. 18, 1934

E. S. ABBOT, M.D., *Presiding*

INTRACRANIAL HEMORRHAGES AND ASSOCIATED LESIONS, WITH SPECIAL REFERENCE TO SUBDURAL HEMORRHAGES. DR. TIMOTHY LEARY.

Forty specimens illustrating extradural, subdural and subarachnoid hemorrhages, and contusion and laceration of the brain were presented. By means of photographic and photomicrographic enlargements, the differences between the subdural space and the serous cavities and the method of production and further evolution of subdural hemorrhages were brought out.

Subdural hemorrhages are relatively common, fifty cases having come to autopsy in the southern medical examiner district of Suffolk County within less than four years. The common cause of so-called spontaneous subdural hemorrhage is the rupture of a bridging vein or a vein on the surface of the arachnoid, usually following traumatism, which may be minor. Death may follow rapidly if a large vein is ruptured. In most cases the blood collects slowly, and symptoms other than headache may be delayed. Temporary hemiplegia may occur. More distinctive is spastic quadriplegia. The lesion is unilateral in over 90 per cent of the cases seen.

If death occurs rapidly the space contains fluid blood (stage 1). The brain shows compression and molding on the affected side by the blood, while the other hemisphere is flattened against the skull. If the patient lives a few days, clotted blood is found in the space (stage 2). If he lives longer, up to two or three weeks, a brown clot and brown bloody fluid occupy the space. In the later period of this stage a definite membrane forms, lining the dura (stage 3), and the clot may be adherent to it. The formation of this neomembrane from the dura is an effort to organize and remove the clot. The arachnoid takes no part in this attempt at repair. If the patient lives several weeks there forms, in addition to the layer of neomembrane lining the dura, an inner layer which envelops the clot and separates it from the arachnoid (stage 4). As a result of the poor circulation to the neomembrane from the relatively avascular dura, the organization of the clot is a slow inefficient process. Particularly is there interference with the venous drainage from the vessels of the neomembrane. As a result of passive hyperemia, the newly formed vessels are overdistended with blood, and widely dilated, sinusoid-like capillaries, known as giant capillaries, are produced. These thin-walled structures, poorly supported by the loose-textured connective tissue, tend to rupture, and secondary hemorrhages within the neomembrane result. These secondary hemorrhages may call attention to the lesion by a dramatic change in the clinical picture and may produce death by rapidly increasing the intracranial pressure. In certain cases, healing of the lesion occurs by fusion of the two layers of the membrane (stage 5), but this end-stage is reached only after months or years. The long continued processes tend to be associated with mental deterioration. The discovery of this stage in persons who have died following psychoses suggests that subdural hemorrhage may be a means of precipitating psychoses.

The only sure method of diagnosis is by exploratory operation. The arachnoid is invaded by histiocytes carrying blood pigment, and this pigment, set free in the cerebrospinal fluid, stains the fluid (xanthochromia). The only method of cure is by operation, which is as much indicated in this condition as in extradural hemorrhages.

The frequency with which this relatively common lesion is overlooked clinically, even by careful, trained observers, is manifest in the record of many of the cases studied.

DISCUSSION

DR. T. J. PUTNAM: Dr. Leary has given a most instructive display of pathologic material. Of particular interest is his demonstration of the development of subdural hematoma. He has shown clearly for the first time that the primary clot is formed by a hemorrhage from a vein crossing the subdural space. It seems astonishing that the arachnoid takes no part in organizing the clot. Perhaps this is because there is a real distinction physiologically between the vascular pia and the vascular arachnoid, or perhaps because of the presence of a small amount of fluid over its surface. The growth of the internal limiting membrane from dural fibroblasts is amazingly rapid, doubtless because it affords an ideal medium for tissue culture. Gardner has shown that the primary increase in volume of the clot is to be attributed to its increase in osmotic pressure, and Fremont-Smith has pointed out that this is doubtless a result of the breakdown of large protein molecules into a vast number of small ones. The extraordinary size of the capillaries is probably due to the fact that the granulation tissue grows under negative pressure, and this may also account for their rupture and the formation of layers of hemorrhage.

The clinical diagnosis is extraordinarily difficult to make. No symptom or syndrome is constant. The intracranial pressure may or may not be increased. There is often xanthochromia of the fluid, but the presence of more than a few red cells practically excludes the possibility of either subdural or extradural clot. The only exceptions to the rule are the cases of extremely severe widespread injury, in which the clot plays a minor part. The only sure way to make the diagnosis is to trephine in all doubtful cases. This should be done, not at the usual site for ventricular puncture, for the clot may not extend so far mediad, but under the temporal muscle—a miniature decompression.

DR. H. R. VIETS: Epidural hemorrhage appears to be comparatively easy to diagnose, from the clinical point of view, and yet Dr. Leary has shown that the diagnosis is not always made except post mortem. One of the chief difficulties in making the clinical diagnosis is in obtaining an adequate history of the onset of the illness. Frequently a patient enters a hospital in coma, and no one accompanies him who can give the history of the accident. On account of this it seems possible that there always will be a certain number of patients who die without a definite diagnosis having been made. Clinically, the diagnosis of subdural hematoma is much more difficult, and many mistakes must be made on account of the nature of the lesion, which is not in a position to give localizing signs. The use of small burr holes through the cranium, with inspection of the subarachnoid space, is a sound way of making the diagnosis and should receive wider recognition. The question of aneurysm as a cause of intracranial or subarachnoid hemorrhage is one of great interest. Aneurysms are being found frequently now, largely as the result of more careful investigation of the state of the cerebral vessels. Many years ago, Dr. James B. Ayer Sr. made some splendid drawings of the cerebral vessels, showing that aneurysms are not uncommon if carefully sought. I wish Dr. Leary would say a word or two more about weak spots in the cerebral vessels where aneurysms are likely to occur.

DR. M. H. A. EVANS: I wish to ask Dr. Leary if there is any clinical sign of laceration of the brain as apart from the rest?

DR. A. W. STEARNS: Most of the cases in which the patients survive result in litigation; I generally see the patients for lawyers from two to five years after the injury. Hospital records of neurologic examinations are usually very inadequate. These patients are rarely seen by neurologists, but fall into the hands of surgeons, who fortunately are usually satisfied to let nature take its course.

DR. TIMOTHY LEARY: After all, the pathologist has the last look. I am not at all critical of failures in diagnosis in this difficult field. I have been impressed with the thoroughness of the study of cases in the neurologic and neurosurgical services at the Boston City Hospital and the response to demonstrations that subdural hemorrhages are common and that operation offers the only method of relief. The difficulty in diagnosis is being met by the exploratory burr hole operation, which is without after-effects. The burr holes will disclose these hemorrhages almost constantly. The location of the hematoma over the convexity of the brain, its usual unilateral character and the fact that it does not ordinarily recur after operation are remarkable. With reference to secondary hemorrhages within the neomembrane, the interference with venous drainage through the dense dura is apparently responsible for the overdistention of what would be ordinary capillaries in a granulating lesion in other locations. The giant capillaries can be followed as such almost up to the point of emergence of the veins in the dural openings. Some with thicker walls than capillaries belong evidently on the arterial side. As one views these dilated capillaries, the wonder is not that secondary hemorrhages arise from their rupture, but that they do not occur more frequently. With reference to aneurysms of the basilar vessels as a cause of massive fatal subarachnoid hemorrhages, the most careful search in some cases will disclose no true aneurysm. Studies of the arteries in the base of the brain have disclosed frequently faulty formation of the media, which varies markedly in thickness and is believed to be the cause of hemorrhage and also of the formation of aneurysms.

Aneurysms of cerebral vessels are usually not of syphilitic origin. In answer to Dr. Evans' question, in my experience contusion and laceration of the brain should be accompanied by blood in the cerebrospinal fluid. It is within the realms of possibility to have a surface contusion of the brain without any tearing of the pia-arachnoid, but I do not think that I have ever seen a case. If the arachnoid is torn, blood will escape into the fluid. Its amount will depend largely on the extent and character of the laceration. With reference to Dr. Stearns' remarks on end-results, I could have brought here a series of brains showing evidence of old traumatism. Epileptic seizures are the commonest sequelae clinically. It is probable that there is a lowering of the mental level in many cases. There are striking exceptions, however. I remember the case of a business executive who was found dead, having fallen forward with his head in a wastebasket and suffered asphyxiation in that position. His history indicated that he had had epileptic seizures. He had been in Boston for several years. Postmortem examination disclosed a large scar in the right temporal region. As the scalp was being dissected from the skull, masses of lead appeared embedded in scar tissue. When the head was opened the right squamous bone was found to be made up of masses of comminuted bone which had been fused together by new bone formation. The anterior half of the right temporal lobe and the inferior portion of the right frontal lobe were made up of a series of cysts. It was evident that an attempt at suicide, a pistol with old ammunition having been used, had failed, the damage to the skull and probably to the dura giving free decompression. The man's colleagues stated that he had been a brilliant business executive.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 18, 1934

PERCIVAL BAILEY, M.D., *President, Presiding*

LYMPHOBLASTOMA OF THE SPINAL CORD. DR. A. ETTLESON (by invitation).

History.—A physician, aged 44, had been well until eight weeks before admission to the Research and Educational Hospital of the University of Illinois. On Dec. 10, 1932, he was seized suddenly with a sharp burning pain in the small of the back. He thought that the pain was due to a lumbar myositis which he believed resulted from sleeping with insufficient bed covering. In an attempt to obtain relief from the pain, the patient took large doses of anodynes and analgesics, had his back massaged and strapped with adhesive, exposed his back to the infrared lamp and took several diathermy treatments. Finally, he resorted to the use of morphine. He was unable to sleep without the use of this drug. In spite of these measures, the pain grew steadily worse until the back felt "as tender as a boil," as the patient described it. The pain began in the middorsal region and radiated in a zone about 4 inches (10.16 cm.) wide across the abdomen, where it felt like a constriction band. It was aggravated on bending forward, by physical exertion, on changing the position in bed, by coughing, by straining at stool and especially when lying flat on the back. It was diminished when the patient sat in a chair with his back not in contact with anything. Whenever the patient coughed he felt a stabbing sensation from the upper end of the sternum to the umbilicus. Twenty-four hours before admission to the hospital, on Feb. 10, 1933, the lower extremities suddenly became completely paralyzed. Within a week, bladder and rectal control were lost, with retention of urine and feces, followed by abdominal distention, impotence, spontaneous contraction of the legs and the appearance of mass defensive reflexes.

There was no history of injury to the spine. Seven years before, the patient had had influenzal pneumonia followed by an empyema. At that time, while

paracentesis in the tenth right intercostal space was being done, the aspirating needle was broken off and could not be located. Therefore, when the patient was examined several days after the onset of the pain, pleuritis, pleurodynia and abscess around the needle were given first consideration in the diagnosis. Dr. George B. Hassin, who was called in consultation, made a diagnosis of compression of the spinal cord, probably by a tumor.

Examination.—There were essentially: complete paralysis of the lower extremities, with spasticity of the right limb but flaccid paralysis of the opposite side, and without atrophy or trophic disturbances; impaired sensation to pain and temperature below the level of the ninth thoracic dermatome, but normal tactile and position sense throughout and absence of any hyperesthetic zone; loss of all the abdominal, cremasteric and suprapatellar reflexes; diminished patellar and achilles jerks, with absence of pathologic signs, clonus, etc.; tenderness on pressure over the eighth, ninth and tenth thoracic spines, and incontinence of urine and retention of feces. The Beevor sign could not be tested as the patient was unable to cooperate. Lumbar puncture showed a greatly reduced pressure of the spinal fluid, the presence of a complete block as evidenced by the Queckenstedt test, xanthochromia and an increase in the cell count, globulin and total protein. The upper extremities, cranial nerves and mental condition were normal. The blood picture was normal; no abnormal cells were found in the blood. Roentgenograms of the thoracic spine showed no abnormalities. The Wassermann reaction of the blood and spinal fluid was negative.

The preoperative diagnosis was extramedullary tumor at the level of the ninth thoracic segment, affecting mainly the right half of the cord, probably of a malignant character, as indicated by the rapid course.

Operation.—On Feb. 13, 1933, Dr. Eric Oldberg performed an emergency laminectomy, removing the laminae of the fifth, sixth and seventh thoracic vertebrae. It exposed a tough, moderately vascular, not encapsulated tumor, about 5 cm. long and 0.5 cm. thick, lying outside the dura. The tumor covered the posterior aspect of the dura and extended around the right side almost to the anterior aspect of the cord. It was easily stripped and was completely extirpated. The point of greatest attachment of the tumor to the dura was at the ninth root on the right side. On opening the dura, the cord was found to be normal but the subarachnoid space was obliterated. No obstruction was encountered on inserting a soft rubber catheter 10 cm. cephalad and caudad.

Course.—The patient voided urine on the day after operation; the pain in the back ceased, and eight days after operation a lumbar puncture showed normal pressure and no block. On the ninth postoperative day, the patient began to suffer from sudden painful contractions of the legs, associated with a feeling of coldness in them, which prevented him from sleeping. He always required that the legs be forcibly pulled on in order to obtain relief. On the eighteenth postoperative day, the first series of deep roentgenotherapy was begun. Three weeks after operation, bladder and rectal control was regained, but motion and sensation in the lower extremities improved only slightly. The patient continued to improve, and ten months after the operation could walk without support; sensation was normal; there was slight stiffness on extension of the right knee. The deep reflexes were moderately exaggerated in the lower extremities, especially on the right, with positive Babinski, Rossolimo and Mendel-Bechterew signs on that side; no pathologic reflexes were present on the opposite side. The cremasteric and right lower abdominal reflexes were absent. There was no adenopathy, and the blood picture remained normal.

Microscopic Examination.—The tumor was a lymphoblastoma.

DISCUSSION

DR. G. B. HASSIN: As Dr. Ettleson demonstrated, the piece removed at operation consisted of vast accumulations of lymphocytes. There were no other cells except a few fibroblasts and a great number of red blood cells. These tumors

have been designated as lymphogranuloma, Hodgkin's disease, lymphadenoma, lymphoma and even lymphosarcoma. In 1932, Viets and Hunter proposed a generic name — lymphoblastoma — for all these apparently heterogeneous tumors because, according to Mallory, the fundamental cells are lymphoblasts, that is, precursors of adult lymphocytes. They also suggested the addition of the subtype; for instance, the so-called acute lymphatic leukemia is classified by Viets and Hunter as lymphoblastoma leukemicum, and Hodgkin's disease as lymphoblastoma, Hodgkin's type. In Dr. Ettleson's case, in which the tumor may be classified lymphoblastoma aleukemicum, we did not suspect the presence of a lymphoblastic tumor, for there were no leukemic manifestations elsewhere in the body. No glands were enlarged except one movable, painless gland in the neck. For this reason it is to be assumed that the tumor originated in, and was confined to, the epidural space, and that lymphoblastomas may occur primarily in the epidural space. What tissues there give rise to these tumors I do not know. Philip Shapiro stated from his brilliant studies that these lymphocytic tumors originate from the epidural reticulolymphatic tissue. If such is the case there must be some factor that causes irritation and proliferation of this tissue. The patient had had various infections, tonsillar, pleuritic and some other thoracic difficulties, and it is likely that some irritative inflammatory process occurred in the epidural tissue that led to the formation of a lymphoblastoma.

I consider this case of great importance, for it shows that lymphoblastomas as they appear in the epidural spaces are probably not real tumors but local reactions of the lymphatic tissue to an inflammatory process.

Optic Connections of the Midbrain and Thalamus. Dr. R. W. Barris (by invitation).

A study of the literature pertaining to the optic connections of the midbrain and thalamus revealed the need for further clarification of concepts of this problem. My colleague and I have therefore studied Marchi-stained sections of the midbrain and thalamus of cats after lesions had been made in the course of the optic pathways.

After unilateral and bilateral enucleation of the eyes, we traced degenerated fibers to the dorsal nucleus of the lateral geniculate body, the pretectal area (nucleus praetectalis) and the stratum opticum of the superior colliculi. We found no evidence of primary optic endings in the ventral nucleus of the lateral geniculate body nor, so far as preparations stained by the Marchi method are concerned, of such endings within the nucleus of the optic tract (nucleus lentiformis mesencephali) or the pulvinar. Neither the anterior nor the posterior accessory optic tracts could be identified.

After removing the major portion of the striate area of the visual cortex we were able to trace degeneration of the corticofugal fibers to the nucleus of the optic tract, the stratum opticum of the superior colliculus (on the same side as the lesions) and to the pontile nuclei by way of the transverse peduncular tract. No striofugal fibers were found to end within the dorsal nucleus of the lateral geniculate body or the pulvinar. Lesions made in the parastriate area of the visual cortex resulted in a degeneration of corticofugal fibers, the distribution of which was identical with that observed after lesions of the striate area.

In order to ascertain whether or not the tectum of the midbrain receives fibers which originate in the dorsal nucleus of the lateral geniculate body, we have placed small lesions in this nucleus with the aid of the Horsley-Clark machine. These lesions were made after a complete degeneration of primary optic fibers of retinal origin following bilateral removal of the eyes six months prior to the time of making the geniculate lesions. We have found no evidence of geniculotectal or geniculopretectal fibers. All fibers the cell bodies of which lie within the dorsal nucleus of the lateral geniculate body are destined for the visual cortex.

A study of pyridine silver-stained sections of the dorsal nucleus of the lateral geniculate body of the cat revealed that fibers of retinal origin establish connec-

tions with this nucleus by means of distinct collaterals which originate as branches from fibers of the optic tract. After giving off these collateral fibers, the optic tract continues on its way to the pretectal area and superior colliculi. This is interpreted to indicate that both "visual" impulses (destined for the visual cortex after synapse in the dorsal nucleus of the lateral geniculate body) and "reflex" impulses (destined for the pretectal area and superior colliculi) are mediated over one and the same fiber of retinal origin.

A study of cresyl violet-stained preparations of the lateral geniculate body of a man who had lost his right eye a considerable time prior to death revealed that crossed and uncrossed fibers of retinal origin terminated in alternate cellular laminae within the lateral geniculate body.

DISCUSSION

DR. PERCIVAL BAILEY: Did Dr. Barris find any evidence of bilateral representation of the lateral geniculate body in both striate areas, and also what is his conception of the function of the pulvinar?

DR. R. W. BARRIS: We did not find any evidence for a bilateral representation of the lateral geniculate body on the cortex. It is an interesting question, and I presume it was asked with regard to the problem of "sparing of the macula" as so often observed in clinical cases of hemianopia. In a case reported by Bramwell, Bolton and Robinson in 1915, that of a patient who had been carefully observed for twenty-five years, there was a complete loss of peripheral vision in both eyes with bilateral sparing of the macula. At autopsy the brain showed considerable destruction of both occipital lobes. The histologic study of the visual areas of both lobes in this case showed atrophy from disuse of the cell bodies in this region, with the exception of cells lying along the lips of the calcarine fissure. The authors thought that this accounted for the sparing of the macula, although they were at a loss to understand just why these cells were more normal than others of the visual area, as the fibers of the optic radiation were affected by the lesion. Their explanation was, as is frequently the explanation, that the macular fibers were more resistant to the toxic influence responsible for the original trauma.

In view of the fact that the fibers of retinal origin reach the cell bodies of the lateral geniculate body by means of collateral fibers from the main optic tract, the primary fiber passing onward to the tectum, it seems to me possible that one and the same fiber may subserve both "visual" function (related to the visual cortex after passing through the lateral geniculate body) and "reflex" function (related to the pretectum and tectum proper, the superior colliculi). Since in lower vertebrates (fishes, amphibians and reptiles) the "visual" function is dependent entirely on the tectum and has no relationship with the primitive cortex, it seems possible that in the case of macular vision one might be dealing with the preservation of this phylogenetically old function presided over by the tectum, and that in the absence of the occipital lobes the sparing of the macula could thus be accounted for. Anatomically, this is a real possibility in view of the joint termination of a single retinal fiber in both the tectum and the lateral geniculate body. Poljak and Putnam have shown that there are no branches of fibers from the optic radiation that cross in the corpus callosum, and thus one cannot account for the sparing of the macula on the basis of a bilateral representation of the macular fibers of the visual cortex of both occipital lobes. The question needs more study, of course, and I offer this solution to the problem merely as a suggestion, which to me seems plausible, though without established proof.

LATE INFANTILE AMAUROTIC IDIOCY: CLINICAL AND ANATOMIC REPORT OF A CASE WITH MARKED CEREBRAL ATROPHY. DR. RICHARD RICHTER and DR. ARTHUR H. PARMELEE.

This article will appear in full in a later issue of the ARCHIVES.

ENCEPHALOMALACIA IN INFANTS. (VIRCHOW'S INTERSTITIAL ENCEPHALITIS.) DR. I. B. DIAMOND.

This article was published in full in the June issue of the ARCHIVES, p. 1153.

DISCUSSION

DR. ARTHUR WEIL: As to Virchow's encephalitis interstitialis neonatorum, I think that one of the best papers on the subject in the recent American literature is the one by Schwarz, Goolker and Globus, published in the April 1932 issue of the *American Journal of Diseases of Children*. The authors go back to the original source of Virchow's description. As I understand it, he described as encephalitis the accumulation of numerous small round cells with dark-staining nuclei in large foci, which erroneously were regarded by him as lymphocytes but which are now recognized as foci of gliocytes. They are most frequently found in the subependymal regions and in different parts of the gray and white matter of the cortex in nearly every new-born child. According to the present prevalent idea, they have no pathologic significance; the demonstration of small particles staining with sudan or myelin sheath stains has suggested that they carry lipoids which, perhaps, may have some connection with the process of myelinization. What Dr. Diamond described suggested to me simply islands of softening, the origin of which I could not guess from his description. I suggest that he should not give to the pathologic picture which he described the name of "Virchow's encephalitis interstitialis," but rather should call it "multiple softening of the brain."

DR. G. B. HASSIN: I think that Dr. Diamond gave the correct interpretation of Virchow's description of what he called "interstitial encephalitis." Virchow published two articles on this subject, but in neither did the description by Virchow justify a diagnosis of encephalitis. His views, as well as those of Yastrowitz (1871), have only historical interest. The gitter cells found in a so-called normal child or in any other person are not builders of myelin and are always pathologic, especially when in large numbers. Such views promulgated in the sixties and seventies of the last century can, as said, be only of historical interest. The case Dr. Diamond described strongly suggested some toxic-infectious process which produced multiple foci of softening. These observations do not support the contention of Schwartz, who published a large monograph, that the changes are always traumatic.

DR. I. B. DIAMOND: I wish to call attention to the fact that the title of my paper is "Encephalomalacia." In Virchow's article there are pictured typical gitter cells which suggest areas of softening of the white substance. He interpreted these foci as fatty metamorphosis of the glia cells.

Book Reviews

Insanity as a Defense in Criminal Law. By Henry Weihofen. Price, $3.75. Pp. 524. New York: The Commonwealth Fund, 1933.

The problem of insanity, one of the most equivocal in American civilization, has its legal and medical aspects. The present volume deals chiefly with the legal aspect and seems to cover the field adequately; at the same time, it rounds out implications of extreme importance to the psychiatrist. It is based on a study made at the University of Chicago with the aid of a grant from the Commonwealth Fund. This study consisted of an exhaustive research into the law as it relates to insanity. The early chapters summarize the status of the tests for insanity, a subject that has been rehashed in every book on the law of insanity. The usual questions are presented: for example, how to determine whether the accused is sane, whether there can be delusions without general undermining of the mental condition of the accused, etc. The New Hampshire rule that there is no legal test for insanity is emphasized. (This rule has not been followed in other states.) The digest of cases following the chapter on tests makes it clear that jurists have no great understanding of the medical problems arising from suspected mental disease. Only one suggestion (the New Hampshire rule)—that the determination of mental illness remain in the hands of medical experts—has met with much encouragement. To those who are engaged in the problem of determining legal insanity a hard and fast test, such as the application of the McNaghten rule, is of no help. The question immediately arises: Whose responsibility is it to show that the defendant is insane? And in the chapter concerned with this phase of the problem the digest of cases shows as many opinions or at least as many reasons for the opinions as there are states. It is interesting to note in this connection that, while reference to specific forms of mental disease is made in the medical literature, the law seems indifferent to the details of the specific disease from which the defendant may be suffering. Yet every physician knows that the responsibility of the patient varies greatly according to whether he is suffering from hypophrenia or from Korsakoff's or Ganser's syndrome.

The question of "expert testimony" is one which has caused the psychiatric profession to fall into disrepute with the public. The sight of one group of medical men testifying for, and another against, a person on trial on the same set of facts is deplorable, but in this author's digest of cases bearing on the subject the reason for this can be plainly seen. The necessity for the expert to be properly qualified is in some states so ignored that even laymen may qualify, and in most states it is not necessary for the physician to show that he has ever seen a patient with a psychosis. He may be called to the stand to express an opinion even though he may have observed the defendant only in the courtroom or in jail; he may have been refused the opportunity for making a thorough study of the defendant, which newer developments in psychiatric criminology have shown to be so necessary for an intelligent understanding of the case. A few laws have been passed which point out a means of correcting this situation—laws which provide for the commitment of the defendant to an institution for observation (in four states) and laws (in six states and possibly in a seventh) which provide for the appointment by the court of experts who shall be independent of those of either the defense or the prosecution. One of the outstanding points for criticism of the whole method of dealing with the problem of insanity is the hypothetic question, on which so much stress has been laid. It is obvious that, since the form of the question must be designed to bring out a specific point, answering the question lies purely within the realm of dialectics and not of medicine. Some interesting facts are presented to show how courts have permitted nonexperts to

express their opinions of the sanity of defendants. The author states: "Psychiatrists have insisted that laymen are incompetent to form a sound judgment as to another's mental condition, even though they are intimate acquaintances. In a few cases this view seems to have been accepted, but such cases are rare." As for the type of evidence which can be admitted, the opinion of the courts regarding medical entities is found to be more or less consistent with the opinion of experts of the medical profession.

A large amount of space in the book is devoted to a discussion of pleading and court procedure, the finding of mental irresponsibility and the release of the defendant on recovery. Here the law treating of insanity at the time of the criminal proceedings is revealed. The final chapter is a summary of suggested reforms. It is conservative; it summarizes only what has been done in the way of preliminary sorting of mentally deranged offenders and the attempts which are being made to revise the tests for irresponsibility. The first of these summaries treats of the psychiatric laboratory and clinic, and reference is made to the education of judges by these means. The author refers to the Massachusetts plan and gives the first summary of that plan which has been made by a writer not in the Massachusetts Department of Mental Diseases. Weihofen seems to think that revision of the tests is necessary, but suggestions which have been made so far do not offer a solution for the problem of what constitutes legal insanity.

In the minds of physicians who are confronted with this medicolegal problem of legal insanity, the question arises whether it would not be a good idea to take much of the power of the courts away from them in this matter. For many years the courts have been inadequate and incompetent. In the concluding pages, Weihofen, who is a lawyer, does not go this far, but he agrees with the psychiatrists that experts should be better qualified, that the jury's function should be abolished so far as scientific questions are concerned and that the law should be entirely revised on the subject of "responsibility" and treatment of the offender. These brief references are the only ones in this thick book in which treatment is mentioned. While the conflict of opinions, the inadequacy of law-making bodies and the stupidity of judges when dealing with insanity are striking, still more so is the conflict of the archaic machinery of the law with the scientific, humane attitude of psychiatric criminology. It is admittedly not within the scope of the work under discussion to bring out the fact (ever present to the psychiatrist) that the line between sanity and insanity is not nearly so important as the question of preventing crime and curing the criminal. Just as the dinosaurs and mastodons became too cumbrous to survive, the organism of the law is becoming so unwieldy that it is beginning to work against its own existence. Nowhere, except possibly in Glueck's "Mental Disorder and the Criminal Law," has this been more clearly demonstrated than in the present work. Weihofen makes it clear by implication that the law no longer considers itself the agent for the social good, but rather the mechanism for justifying past ideas, even though their validity and significance are out of date. The contempt of the law for scientific opinion and its inability to devise a hierarchy of competent experts are apparent and point the way to the beginning of reform. If critical and politically potent law-makers can be found, it would be well to put this book in their hands and call their attention to the following factors: the inconsistency of the law in the various states; the failure of the law to provide for competent advisers to the courts, and its failure to furnish safeguards for society and psychiatric treatment for the offender. After all, the decision about a man's mental condition is not a mere dialectic game, but a thing of great moment to himself and to his neighbors. Like all textbooks on law, this one is profusely filled with citations of cases and references to literature. In this respect, it must be remarked, that the references to medical literature are inadequate, and that out-of-date opinions are given as much importance as more valid ones. As for the legal decisions, a case is just a case. A good bibliography is appended. Physicians who have to testify in cases of insanity will find this book useful in clarifying their duties and limitations. Mental hygienists should find

it a worthy tool for reform. Opinions which the reviewer has obtained from lawyers would indicate that they, too, consider it a praiseworthy piece of work, and one hopes that it will give the law insight into its own unjustified self-sufficiency.

Lying and Its Detection. By John A. Larson, in collaboration with George W. Haney and Leonarde Keeler. Price, $5. Pp. 453. Chicago: University of Chicago Press, 1932.

This volume presents a survey of the various methods which have been employed in ancient and modern times to detect deception, together with a description of the technic which the author has developed. His "lie detector" is a cardio-pneumographic record of the suspected person, taken while the latter is answering a series of direct questions the implications of which he must either affirm or deny. The graphs are obtained by a specially devised sphygmomanometer.

The first section of the book deals with the definition of lying and the types of lying, and the second, with the methods that have been used to determine guilt or innocence from early times to the present. The difficulties involved in all these, from the methods of torture to the modern judge and jury system, are shown by quotations from criminologists and other investigators and by reports of illustrative cases. In the third section the author reviews modern scientific methods—experiments in word association and reaction time, records of breathing, galvanometer tests and the measurements of blood pressure made by Marston and others. The material in these three sections is of value for reference, but it is presented chiefly by series of abstracts from various writers and consequently makes difficult reading. Although the author relates the different opinions and inserts critical comments, he leaves to the reader much of the task of unifying the problems discussed.

In spite of the large amount of data in these three sections, readers with psychiatric or psychologic interests will wish that Larson had gone further into some of the problems, particularly those in connection with the nature of lying and the psychologic processes involved. His work differs here from the recent work of Luria in Russia, an account of which was published in this country two years ago under the title "The Nature of Human Conflicts." Larson's greatest interest is in the practical aspects of the problem of lying and the detection of lying, while Luria's interest was in analyzing behavior and mental organization.

Both Larson and Luria made their experiments on persons who were suspected of crime. Their work is thus a great advance over most previous studies of lying, in which both the penalties and the rewards were limited to the university laboratory. Incidentally it may be noted that Larson suggests the use of his technic not only with suspected criminals but also with psychiatric patients. He reports that by the present technic, that is, a general questionnaire and the polygraph, it has been possible to discover and unravel complexes which have not come to light in extensive psychiatric examinations.

In an early chapter the author discusses the physical concomitants of emotion and surveys some of the leading views, but he does not attempt to take up controversial subjects.

As to the immediate rôle which the lie detector is suited to play, Larson gives a definite word of warning. He states: "Up to the present time the writer has been strongly adverse to the introduction of deception-test records into court procedure. . . . There can never be in this technic such accuracy as is possible in the case of finger-prints, and so long as error is possible, these records should not be introduced for wrangling sessions by opposing attorneys. . . . But as a means for the interrogation of suspects and their accurate selection, it compares favorably with other police or legal methods of investigation, if used properly, and has already won an important place for itself as a tool in preliminary investigations."

The layman who is interested in criminal procedure, as well as the specialist who has to deal with suspected persons or witnesses, will find the book of great

interest and value. The survey of the legal, police and scientific methods of investigation outlines the situation today and indicates the practical and theoretical problems which are greatly in need of further study.

Our Neurotic Age. Edited by S. D. Schmalhausen. Price, $4. Pp. 531. New York: Farrar & Rinehart, 1932.

The contributors to this symposium have been drawn from several different professional fields, including those of psychology, psychiatry, economics and sociology, anthropology, philosophy and literature. A natural result is that the chapters present varied points of view and opinions. The approach is sometimes in terms of the individual patient and of the psychoanalytic theory, and at other times from the view that present civilization, particularly because of its economic organization, furnishes a setting conducive to mental maladjustments. The editor's introduction leaves little doubt that he sympathizes with the second attitude; perhaps one reason for his sponsoring such a volume was the desire to bring home to the reader that this is a neurotic age because of the social forces that affect the persons living under them. Yet he has not selected the contributors solely from those who are concerned with fixing the responsibility for the increasing quota of the mentally maladjusted on the present social and economic order, but has seen to it that those who stress the rôle of personal relationships and emotional development in the causation of mental abnormalities have an equal representation.

There are twenty-seven chapters by as many different authors; even a partial list of these suggests something of the nature of the volume: John J. B. Morgan writes of "The Mind in the Breaking" from the point of view of educational psychology; Ernest Jones gives a psychoanalytic concept of the normal mind; Jastrow treats of "The Maladies of Thought"; Roback considers the question "What is Sanity?" Among the chapters on special topics in the field of abnormal psychology are those on suicide, homosexuality, alcoholism, etc. Certain social phenomena which have occupied public attention are given a place in the contributions on nudism and racketeering. Ira Wile has a chapter on "Problem Parents and Problem Children." Calverton reviews the trends in modern literature as reflecting the pathology of modern culture.

It is interesting to note that along with the considerable difference in point of view among the contributors (except within the group who agree that the economic and social order in this country is fundamentally unhealthy) there is some inclination manifested by almost all of them to indulge in prophecies as to the future or to outline remedies for existing conditions. The same variation is apparent in these prophecies and plans as in the other matters. The book therefore becomes somewhat of an open forum, and presumably the reader is free to choose between the diverse opinions which have been offered.